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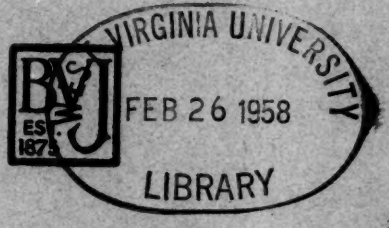
A Monthly Review of Veterinary Science



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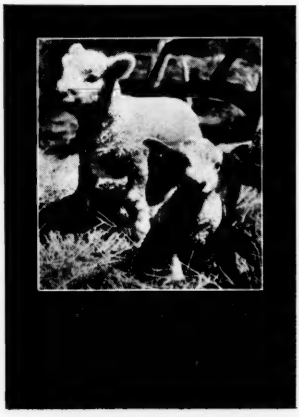

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The British Veterinary Journal

JANUARY, 1958

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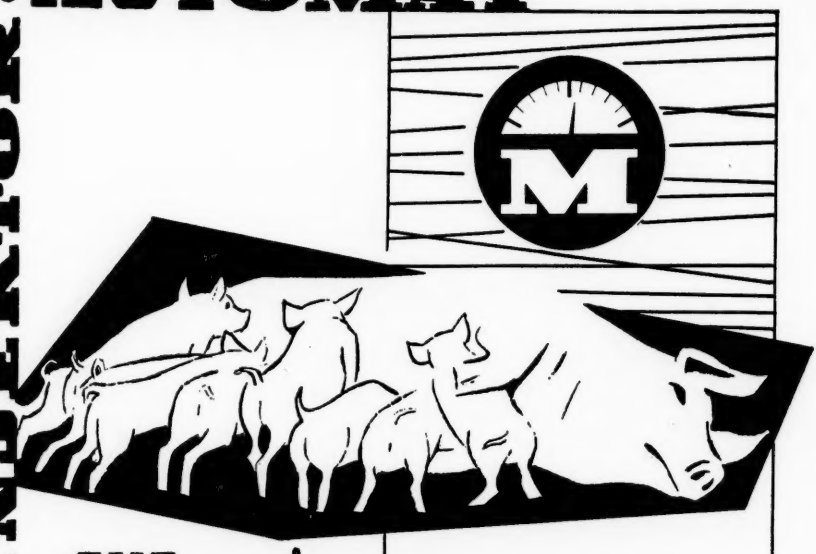

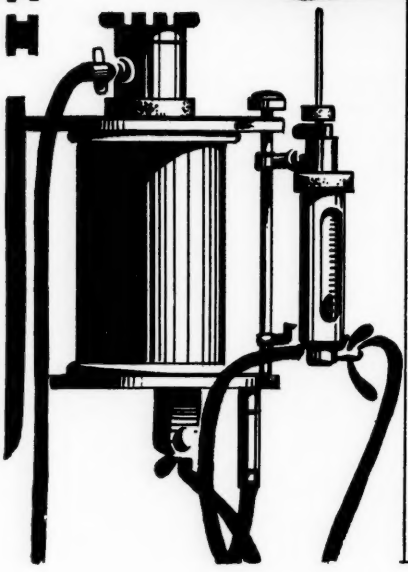

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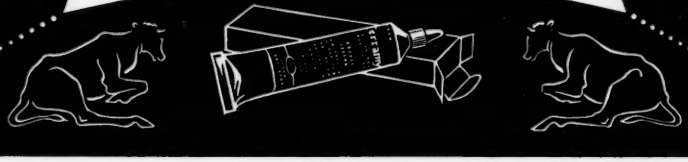

THE BRITISH VETERINARY JOURNAL

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EDITORIAL

GOOD NURSING

"I am deeply distressed to note that modern veterinary surgeons treat their cases in a purely factual medical or surgical aspect and seldom, if ever, offer any advice to the owner in respect of nursing".

This is an extract from a letter which was addressed to one of the Editors by a very experienced and competent Kennel manager. Similar views have been expressed elsewhere as regards the medical profession. It would seem that surgical and medicinal therapeutic measures have attained to such a high standard of precision, specialisation and efficacy that the general care and nursing of the patient, which was so important in more primitive times, is considered to be of little consequence. Those who have entered the healing professions in recent years, in the thrilling times of antibiotic and chemotherapeutic discoveries, are the greatest offenders. The modern concept seems to be, a diagnosis has been made, it must be correct for innumberable ancilliary aids have been called into service, the most recently discovered remedy has been applied, it must have a specific action for it has a long name containing many letters and symbols, so the doctor's duty is completed and the patient can be left to seek his own salvation.

It has been said that, even after major operations, the modern surgeon is not greatly interested in his patient, when the latter has left the operating theatre. It would seem that the present attitude is not encumbered by any humanitarian concept. A precise repair has been made so why worry, the patient cannot avoid a full recovery. This type of outlook is not confined to the medical and veterinary professions it has pervaded into many walks of life. The expert mechanic who tunes a carburettor or adjusts the electrical system of a car so that the engine purrs like a contented cat will wave one joyfully out of the garage door and ignore the tyre that is flat on the near hind wheel. Animals and men did recover from serious diseases

before the days of antibiotics and the sulpha drugs. We know that many of the medicinal agents used in those times could have little if any influence on the process of repair and probably their greatest quality was that although they did not specifically advance or stimulate the return to normal they did not unduly interfere with the natural defensive mechanism of the animal body. With very few exceptions successful recovery from an illness or injury depends in great measure upon the patient's inherent ability to resist assault and that superb quality "the will to live and survive". In the veterinary sphere the best examples of these attributes can be observed in the dog full sick with distemper or a cat which falls victim of the "so called" feline influenza. These are two occasions in which good nursing is a potent factor, surpreme above all others, in the promotion of recovery. A gentle caress and the smoothing of a pillow or cushion can reap more reward than a hypodermic injection. Nursing is an art and a science and those who prescribe its form should be competent and experienced practitioners. The only way to attain proficiency is by virture of actual practice. Veterinary and medical students should serve a period when they are in actual hour to hour charge of patients who are ill. They should learn how to bed, feed, clothe, talk to and attend to the hygiene of their patients so that they may be in a position to give instruction to others and if necessary demonstrate by precept.

It would be ridiculous to presume to give instructions for the preparation of a boiled egg if one did not know how to boil water. It is well to remember that a soothing hand can engender the will to fight and live and good nursing has saved more lives than any drug. If these gracious things are combined with the wonderful modern therapeutic agents which are at hand the patient's chances of recovery can be enhanced in very great measure.

VACCINATION OF CATTLE WITH B.C.G.

by T. M. DOYLE and P. STUART

Central Veterinary Laboratory, Weybridge

In view of the campaign that is being carried out by the World Health Organisation with B.C.G. against human tuberculosis, the results of its use in a herd of dairy cattle which contained many reactors may be of interest. The herd consisted of 65 pedigree shorthorn cows and about 85 young stock; it was self contained apart from the occasional purchase of a bull. A tuberculin test in 1934 revealed about 80 per cent reactors in the milking herd and a considerable number among the young stock. One group of 15 heifers one to two years of age were sold because many of them were reactors. Two further tuberculin tests were made some years later by two different observers. Tuberculins of different origin were used and again a large number of reactors were found. There were some discrepancies between these tests and the original one, but all showed the herd to be heavily infected. The discrepancies could be explained by a change in the tuberculous status of animals with the passage of time and the use of different tuberculins. There were considerable variations in the potencies of the tuberculins in use at that time. An attempt was made to build up a clean herd by separating reactors from non-reactors, but this was impracticable under the conditions prevailing and was discontinued.

TABLE I
Tuberculin tests made in 25 vaccinated cattle.

	Negative at post-mortem examination	Positive at post-mortem examination
Number of animals	15	10
Mean mammalian excess (mms).	6.03	15.00
Range of excess (mms).	0-10	7-40
Doubtful group (7-10 mms. excess)	7	4
Standard deviation	± 3.08	± 10.24
Limits (mms.) P = 0.2	4.96 - 7.10	10.56 - 19.44
Limits (mms.) P = 0.3	5.17 - 6.89	11.46 - 18.54

In 1943 the owner consulted this laboratory. He was unwilling to dispose of the herd and replace it with clean stock as the average milk yield was high and represented many years of careful selection and breeding. To have eliminated the reactors would have been tantamount to the disposal of the herd. Although undoubtedly the infection was widespread the herd had always been in good condition and during the years it had been under observation three clinical cases only had occurred. Having regard to these facts the owner was advised that the only practical method of dealing with the situation was to use B.C.G. It was explained that B.C.G. was innocuous, that it would confer a considerable degree of protection against the gross infection present and that it should be possible over the course of years to build-

up a tuberculosis-free herd behind the protection conferred by the vaccine while retaining on the same premises the infected animals for their normal economic life. In view of the heavy infection in the herd when vaccination started future attestation was not particularly considered, but the rapid progress made in recent years by the Attested Herds Scheme and the establishment of eradication areas altered the position so markedly that the elimination of reactors became of cardinal importance. This proved to be a slow and expensive procedure with B.C.G. vaccinated animals.

Vaccination

Each calf was vaccinated within 10 days of birth, again at 6 months of age and then once a year. The dose of vaccine was 100 mg. in 10 ml. of a Sauton-saline mixture. It was injected subcutaneously either on the side of the neck or into the brisket. There is no accurate information on the duration of protection conferred on cattle by one dose of B.C.G.; estimates on the duration in human beings vary from four to ten years. In view of the heavy infection existing in this herd it appeared that repeated vaccination was advisable.

Feeding of Calves. The calves were bucket-fed. Each received 50 galls. of milk. All milk—other than colostrum—was boiled. This was a laborious procedure but was considered to be essential for the prevention of infection before vaccinal protection was established. A separate attendant was provided for the calves. Young stock were isolated until they entered the milking herd. Then they mixed freely with the non-vaccinated reactor animals at pasture and in open sheds during the winter so there was ample opportunity for infection to occur and the conditions provided a severe test of the vaccine.

Vaccination of calves was started in 1943 and in addition tuberculin negative stock which were two years of age or under at that time were vaccinated also. Vaccination was carried out regularly until 1949 when it was discontinued in the milking herd. For a further two years new born calves were given a dose of vaccine which was not repeated. The last vaccination was carried out in January 1952.

Preparation of Vaccine

When vaccination started B.C.G. was being prepared at the laboratory on Herrold's solid egg medium, for a field trial then in progress. It was from this source that the vaccine was obtained. It soon became evident that B.C.G. grown on this medium was unsatisfactory. In some animals, following the second dose of vaccine, a large fibrous nodule formed at the site of injection. More animals were affected after the third injection and the nodules were larger. Three recently calved heifers in good condition vaccinated for the fourth time exhibited serious shock. Within a few days they went off their milk, became emaciated, and each developed a large oedematous swelling over the upper part of the ribs. These animals remained in poor condition for many months and their milk yield did not return to normal until the next lactation. The cause was apparently foreign protein shock induced by the presence in the vaccine of egg material scraped from the surface of the medium with the bacillary growth. The use of Herrold's medium was discontinued, vaccine was prepared from growth on Sauton's medium in accordance with the method of Calmette and Guerin and no further trouble occurred. If the method of preparation

had not been changed serious losses from shock would probably have occurred among animals vaccinated subsequently.

TABLE II
Tuberculin Tests on Vaccinated Cattle.
Mean Reactions to Avian and Mammalian Tuberculins.

Date of test	No. of animals tested	Mean Reaction	
		Avian	Mammalian
15.11.49.	5	8.80	10.60
8. 7.50.	9	7.17	10.22
2. 5.51.	11	4.62	8.32
5.10.51.	7	3.57	6.83
13. 2.52.	13	1.92	5.62
12. 8.52.	13	3.73	6.88
11.11.52.	13	3.19	4.35
12. 5.53.	13	3.58	5.85
Sept. 53.	13	3.00	4.23
May 54.	13	3.27	5.04
Aug. 54.	13	2.15	2.69
Nov. 54.	13	2.23	1.54
Feb. 55.	13	1.77	1.46
Apr. 55.	13	1.85	1.62
July 55.	13	2.92	2.08
Sept. 55.	13	3.00	2.08

Tuberculin Tests

When many of the original non-vaccinated reactors had been disposed of at the end of their economic life or for other reasons, and when a reduction in the number of infected animals was expected, vaccination of the milking herd was discontinued and one year was allowed to elapse so that post-vaccination allergy might fade before tuberculin tests were made with a view to attestation.

Ten herd tests, using avian and mammalian tuberculins intradermally in the neck were carried out in the period 1951-1954. The degree of reaction is estimated by the difference (in millimetres) between measurements of double skin folds at the site before, and seventy-two hours after, injection. The detection of the infected animal which reacts to both tuberculins depends on the difference between the mammalian and avians measurements, the greater the excess of mammalian reaction the greater the chance that the animal is infected. Since B.C.G. is a bovine strain of the tubercle bacillus some mammalian reactions were expected, but the number (35 per cent) which had a mammalian excess of 7 mms. or more found in the first herd test in 1951, was greater than expected considering that vaccination had been discontinued for one year. The considerable number of reactors created a difficult situation as a decision had to be made whether to leave them in the herd in the hope that the allergy would fade or to remove them in case they were infected.

In the period 1951-1953 twenty-five vaccinated animals with mammalian excess reactions varying from 0 to 40 mms. were submitted to examination post-mortem. The average interval between the last B.C.G. vaccination and slaughter was two years. In fifteen of these no macroscopic lesions of tuberculosis were found and biological tests on pooled lymph nodes were negative. *M. johnei* was isolated from two of these fifteen animals and avian tubercle bacilli from one. Lesions of tuberculosis caused by the bovine type bacillus were found in ten animals, three exhibited generalised infection, four had lesions in the lungs, two in the pharyngeal lymph nodes only and one

had lesions in a mesenteric node only. The range, mean and standard deviations of mammalian excess tuberculin reactions for the uninfected and infected groups are shown in Table 1. Almost half of the animals in the uninfected and four out of ten of those in the infected group lay in a " doubtful " or overlapping range of 7 to 10 mms. Using the limits calculated from this small sample four out of five infected animals might be expected to have an excess of 10 mms. or more, while two out of three uninfected animals might show mammalian excess reactions of 7 mms. or less. Approximately forty per cent of all animals would fall into the " doubtful " class of 7 to 10 mms. By 1954 the number of animals which had an excess reaction of 7 mms. or more had fallen to two (3.1 per cent).

In addition to the twenty-five animals which were slaughtered, twenty-five others were sold because of their reactions to the tuberculin test. The last case of tuberculosis discovered on post-mortem examination was in February 1952 but it is possible that some of the animals sold at a later date might have been infected.

Graphs I and II and Tables II and III show the test histories, for five years after their last vaccination had been carried out, of thirteen cows which had been vaccinated four or five times. It will be noted that nearly five years elapsed before the tests became negative. This persistent sensitivity may have been caused either by multiple doses of vaccine or by repeated re-infection.

During the five years since vaccination was discontinued 100 home bred, unvaccinated animals have entered the milking herd. Six herd tests have been carried out during that period and none of these unvaccinated animals has given a positive reaction. This would suggest that the reactions exhibited by animals in the herd during the period were not the result of natural infection.

TABLE III

Tuberculin Tests on Vaccinated Cattle.

Mean reactions to Avian and Mammalian Tuberculins arranged according to time that has elapsed between last B.C.G. vaccination and tuberculin test.

Time (mths)	Mean Reaction		Time (mths)	Mean Reaction	
	Avian	Mamm.		Avian	Mamm.
1- 3	7.0	10.2	49-51	3.2	3.8
4- 5	-	-	52-54	3.0	5.3
7- 9	5.9	8.7	55-57	2.6	3.4
10-12	6.5	8.0	58-60	2.1	1.7
13-15	2.5	9.5	61-63	2.2	2.1
16-18	4.6	6.2	64-66	1.5	1.5
19-21	1.2	5.0	67-69	3.0	2.0
22-24	3.9	7.1	70-72	2.7	2.0
25-27	2.0	6.1	73-75	1.0	1.0
28-30	2.1	3.4	76-78	2.0	2.0
31-33	2.9	5.5	78-81	1.0	2.0
34-36	2.7	4.1			
37-39	1.9	1.4			
40-42	3.2	4.2			
43-45	3.0	3.7			
46-48	3.2	4.0			

Discussion

The use of B.C.G. on this heavily-infected herd was not a satisfactory test of its efficacy because many positive and doubtful tuberculin reactors were sold to speed up

attestation. Calmette (1927) stated that animals inoculated subcutaneously with 100 mg. or 50 mg. of B.C.G. reacted to intradermal tuberculin as long as the vaccinal lesion could be seen or felt. Two out of seven of his experimental animals ceased to react by the sixth month and only one animal reacted by the twelfth month. Van Deinse (1952) found that in the absence of repeated re-infection the tuberculin sensitivity of vaccinated animals disappears after a time.

In our experience animals given 100 mg. subcutaneously and kept on clean premises rarely react for as long as twelve months; and when vaccine is given intravenously the sensitivity usually disappears about the sixth or seventh month. We could find no information in the literature on the duration of sensitivity in cattle to multiple doses of vaccine; and because of the probability of repeated re-infection in this herd the tuberculin tests provide little information on that point.

The reactions that occurred could have been caused either by vaccination or by natural infection, and there was no way of differentiating between them, as the diagnostic value of mammalian tuberculin was destroyed. The reactions varied widely in size and intensity. In some animals, subsequently proved by examination post-mortem and biological tests to be free from infection, they could not be distinguished from those of natural infection. This varying degree of sensitivity is in accord with observations reported by the World Health Organization (1955):

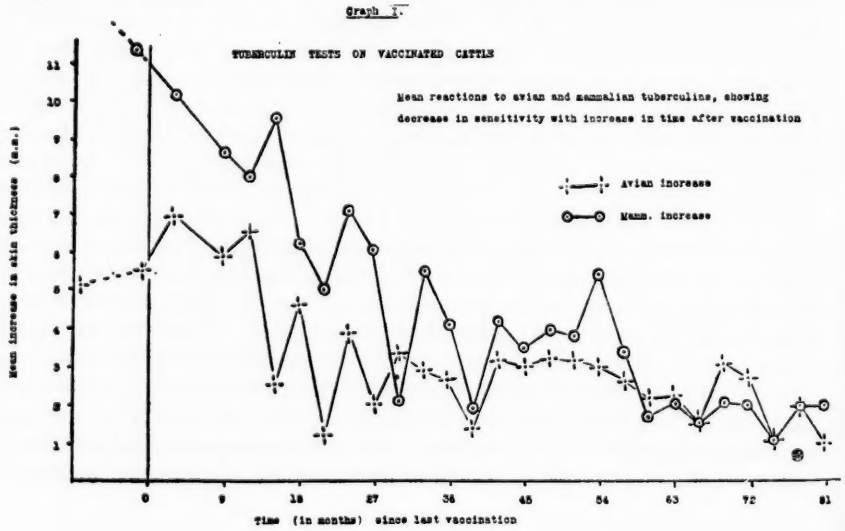
" More critical field studies carried out in recent years (Edwards *et al.*, Mande and Heut, Palmer) have revealed that the kind of tuberculin sensitivity induced by B.C.G. is not a qualitative attribute, that is, one either present or absent, as implied by the terms 'positive ' and ' negative '. Rather, post-vaccination allergy is quantitative, manifesting itself in differing degrees, ranging from barely perceptible reactions to a strong dose of tuberculin to large reactions to a weak dose, from almost no allergy to allergy as strong as that caused by virulent tuberculous infection."

It is generally recognised that B.C.G. does not confer absolute immunity. According to Calmette and Guerin (1929) virulent tubercle bacilli do not circulate in the system of vaccinated animals. From the point of entry they are retained in the nearest lymphatic gland and may form a tubercle, but usually they show no tendency to form extensive lesions; and after a variable period the tubercle becomes isolated, calcified or sclerosed. It is possible therefore that some of the lightly infected, vaccinated animals in this herd might eventually have recovered and become anergic. The presence of active tuberculosis in some of the earlier vaccinated animals was probably the result of the gross infection to which they were subjected, overcoming the resistance conferred by the vaccine. Some infection may also have originated from a five-year-old bull that manifested clinical signs of the disease and was found on examination post-mortem to have the lesions of a severe generalized infection. One side of the bull's pen was formed by the end of a wooden barn in which successive groups of calves, three to six months of age, were housed. There were many interspaces in the partition through which the bull sniffed frequently at the calves. In retrospect it was probably a mistake to have vaccinated some of the original young stock, on the basis of one negative tuberculin test. Milk samples from all the cows, pooled in groups of five or less, were tested, once in 1952 and twice in 1953, for tubercle bacilli by guinea-pig inoculation. All tests were negative. Apart from the infection

present in some of the earlier vaccinated animals, the vaccine appears to have conferred good protection considering the initial gross infection.

There is good evidence (Ascoli, Girard, Gerlach, Hautcoeur and Richart) that tuberculosis can be eradicated in five to eight years from a herd by the use of B.C.G. and the gradual elimination of the unvaccinated stock. The time required for eradication depends mainly on the amount of initial infection and the speed of disposal of the non-vaccinated stock. Brinet (1929) eradicated the disease by vaccination in six years from a herd which previously contained eighty per cent reactors. Govorov (1954) used B.C.G. on farms where tuberculosis was of long standing and difficult to eradicate. The best protection was obtained by two intradermal inoculations one month apart; 4 mg. of dried B.C.G. were used for each injection. Ten calves, vaccinated fourteen months previously were placed in contact for five months with tuberculous cattle. One calf developed tuberculosis thirteen months after exposure. Twenty-seven per cent of the calves reacted to tuberculin up to twenty months after immunization. Rolls and Wiethe (1956) eliminated the disease by vaccination, in seven years from a heavily infected herd. But despite numerous reports on the efficacy of the vaccine it cannot in the light of existing knowledge be recommended, except, perhaps, in unusual circumstances; for example, Vergani (1956) considers that bovine tuberculosis is so widespread on the Lombardy plain that B.C.G. would appear to be the only practicable method of controlling it. When the annual loss of meat, and milk through tuberculosis is taken into account it is clearly more economical to eradicate the disease quickly by testing than by the slower method of vaccination; and there is also the serious public health objection to retaining known tuberculous cows for their economic life as in the vaccination procedure. The value of vaccination as a means of reducing infection before tuberculin testing is nullified by the prolonged sensitivity induced in an infected herd. Having regard to these facts an increasing number of countries are basing eradication on testing and the immediate disposal of reactors.

Graph 1.



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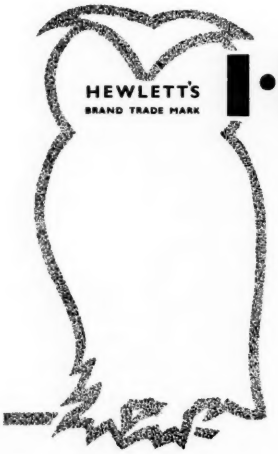

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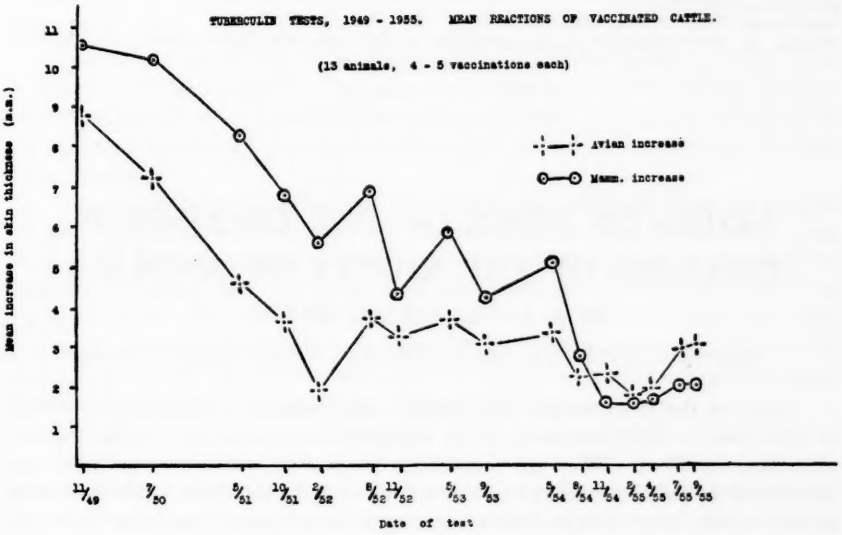

Summary

The use of B.C.G. in a heavily infected herd is described. The repeated inoculation of cattle with B.C.G. grown on Herrold's solid egg medium caused protein shock. In some animals post-vaccination allergy lasted for five years; this may have been induced either by multiple doses of vaccine or by repeated re-infection.

The annual losses from tuberculosis, direct and indirect, are so heavy that when practicable it is more economical to eradicate the disease quickly by testing and disposal of reactors than by the slower method of vaccination.

Acknowledgements

To Miss C. N. Hebert, B.Sc., for the calculations, tables and charts.
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REFERENCES

Ascoli, A. (1948) *Premier Congres Internat. du B.C.G., Paris.*
Brinet, M. P. (1929) *Rec. Med. Vet. 105*, 134.
Bull Wld. Hlth. Org. (1955) *12*, 15.
Calmette, A. and Guerin, C. (1929) *Rev. Gen. Med. Vet. 38*, 385.
Gerlach, F. (1948) *Ibid.*
Girard, G. (1948) *Ibid.*
Govorov, A. M. (1954) Abs. *Vet. Bull. 26*, 555, 1956.
Hautcoeur, P. (1948) *Ibid.*
Richart, A. (1948) *Ibid.*
Van Deinse, F. (1952) *Rev. Med. Vet. 103*, 17.
Vergani, A. (1956) Notiz 1st vaccin. antitubere, *6*, 195. Abs. *Bull Institt. Pasteur*, 1957, *55*, 193.

NOTES ON SOME OF THE UNCOMMON DISEASES OF THE KIDNEY OF ANIMALS

By K. P. CHANDRASEKHARAN

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SOME of the less common and possibly rare diseases of the kidney met with in the course of diagnostic work in the department of pathology over the past six years are described. They are considered because of our own experience and the paucity of published reports. None were detected when the animals were alive either because they produced no clinical signs or if present the latter were not specially referable to the kidney. The main features with some comments on the causation and pathogenesis are presented. These conditions are dealt with more adequately in human pathology and much information has been drawn from this source.

I. Lipomatosis of the Kidney—Cattle

In medical literature a reference to a rare pathological condition known as renal " lipomatosis " has been made by some workers. Though no standard text-books on human pathology mentions this condition, books specially dealing with diseases of the kidney give some information on the subject. Thus Bell (1947) refers to lipomatosis of the kidney " as replacement of renal area by adipose tissue ". Such a kidney is usually enlarged and on mid-sigittal section one finds a thin layer of renal tissue surrounding a mass of adipose tissue and covered externally by a thick layer of fat.

In veterinary literature Edgson (1952) has drawn attention to a condition called " bovine lipomatosis " which he considered to be the occurrence of diffuse over-growth of adipose tissue. He believed that the condition which Gaiger and Davies (1946) described as " diffuse lipoma " in the abdominal cavity of cattle, was similar. Lipomatosis appears to be a diffuse neoplastic condition of the adipose tissue which need not necessarily involve the kidney tissue. When it affects the kidney, the fat

invariably surrounds the organ with practically no infiltration into the parenchyma. On the other hand in medical literature the term as applied to kidney refers to fatty replacement of the renal parenchyma and this replacement is not necessarily a part of a state of generalised adiposity. The condition is not regarded as being concerned with neoplastic process of the adipose tissue.

Two cases are recorded, the first conforming in essential features to the type described in human beings and the other is similar to one of the cases described by Edgson (*loc. cit.*).

Case No. 1 (A.245/50). The subject was a cross-bred heifer calf aged about 3 years and in fair condition. The animal met with an accident resulting in extensive fractures and it was destroyed on humane grounds. At autopsy excepting for the unusual appearance presented by the right kidney no other special features were noticed.

Gross appearance of the kidney: The right kidney was very small. It weighed 50 gms. and measured $8 \times 6\frac{1}{2}$ cms. On sagittal section it was seen that only a thin strip of the renal parenchyma was let and this served to separate the perirenal fat from the medullary fat. The renal pyramids could not be distinguished and were replaced by fat (Fig. 1). The renal capsule was normal and peeled off easily leaving a smooth cortical surface which was pale brown in colour. The calices majores connected with the atrophic cortex by narrow channels and were surrounded by fat. The quantity of perirenal fat was not excessive.

The left kidney was normal, weighed 370 gms. and measured 15×13 cms.

Microscopical appearance of the affected kidney. Sections showed a general resemblance to chronic nephritis. The glomeruli were fibrosed, atrophied and some fused with the surrounding fibrous tissue. A number of tubules were atrophied and replaced by fibrous tissue, while a few showed cystic dilatation. Some of the latter contained hyaline acidophilic casts. The walls of the blood vessels in the existing renal parenchyma were much sclerosed and their lumina very much reduced. There was general increase of fibrous tissue around the glomeruli and tubules and a diffuse infiltration with small round cells.

The intermediate zone was very narrow and fibrosed and from this area columns of fibrous tissue extended towards the hilus enclosing the collecting ducts and papillary ducts. The epithelium of these ducts was 3 or 4 layers deep and surrounded by dense collections of lymphocytes with small islets of macrophages.

The medullary zone was absent being replaced by adipose tissue. The fibrous tissue from the intermediate zone extended into the adipose tissue dividing it into imperfect lobules.

Comment: Obstruction of the urinary passages by calculi has been recorded in most cases of renal lipomatosis in man. Kuttzman (1931) observed this in 26 out of 30 cases. Young (1932) mentioned 11 cases and of these 10 were due either to calculus or to infection, and in one case the cause could not be ascertained. Priestley (1938) observed a case in which the kidney weighed 1,000 gms. Hamn and De Veer (1939) stated that lipomatous change of a mild degree occurred simultaneously with senile atrophy, where there was no calcular obstruction or pyelonephritis and that only obese persons suffered from the condition. Bell (*loc. cit.*) held the view that in the pathogensis of the lesion there was a primary atrophy of the kidney due to such

causes as calcular obstruction, pyelonephritis or arteriosclerosis, followed by replacement lipomatosis. The adipose tissue increased as the parenchyma atrophied and this was derived from that normally present in the renal sinus. The perirenal fat also increased as the condition progressed.

The subject in which the condition had occurred was neither old nor obese, but a young heifer in fair condition. There was no visible obstruction anywhere in the urinary passages. Lipomatosis appeared to have developed as a result of primary atrophy of the kidney brought about by arterio-sclerosis and followed by intra-renal replacement by fatty tissue. The absence of fatty infiltration of the tubular epithelium supported the view held by Young (*loc. cit.*) that fat accumulated only in the renal sinuses surrounding the calyces. The quantity of perirenal fat in this case was not excessive and the kidney itself was not enlarged as was usually the case in lipomatosis. It was possible that these changes might have progressed had the animal lived for some more years.

Case No. 2. Subject: A cross-bred heifer calf, aged about 3 years and in poor condition. The animal was involved in an accident on 27.1.52 and was treated at the College Hospital. On 7.2.52 it died and it was examined post-mortem. No clinical signs were observed while it was alive.

Autopsy (A.55/52). Both kidneys appeared small in size and surrounded by a large amount of compact lobulated masses of fat. The left kidney with the fatty masses weighed 660 gms., whereas the other weighed 420 gms. On sagittal section of the left the fat zone measured 12 cms. across anteriorly and about 8 cms. posteriorly. A separate lobulated mass of fat, $20 \times 15 \times 5$ cms., was present behind the right kidney. No fatty tissue was seen in any other part of the viscera. None of the other organs presented any gross changes.

Histologically, the kidneys showed congestion of the glomerular tufts, varying degrees of degeneration of the epithelium of the tubules and there was granular material inside them. Focal areas of congestion of the intertubular capillaries was observed. On section the fatty masses showed fibrous trabeculæ in between. The amount of fibrous tissue in some areas was great and thus gave the appearance of fibro-lipomata. Many scattered areas of fat necrosis with giant-cell formation and lymphocytic infiltration were seen in the adipose tissue. A well-developed fibrous capsule surrounded the fatty masses.

Comment: The case described greatly resembled the sixth of the series reported by Edgson (*loc. cit.*), but without any lesion in the mesentery or thoracic cavity. Also there was no hyaline changes in the glomeruli or blood vessels. On histological grounds the adipose mass could more aptly be termed as fibro-lipomatosis. The ætiology of the condition is not clear, but Edgson contended that it may be due to disturbances in fat metabolism associated with a genetic or breed factor. However, in three cases in which the cholestrol content of blood was determined, he found no alteration. He considered that the presence of large masses of adipose tissue inside the abdominal cavity might impair fertility and general health. The subject was a heifer in poor condition and considering the size already attained by the adipose masses, it was quite possible that had the animal lived longer, it would have greatly interfered with the normal functioning of the adjacent organs.

PLATE I

FIG. 1
Lipomatous kidney (right) with the normal kidney (left). Note the small size and extensive replacement of the renal tissue by fatty masses. Heifer calf. (A.245/50).

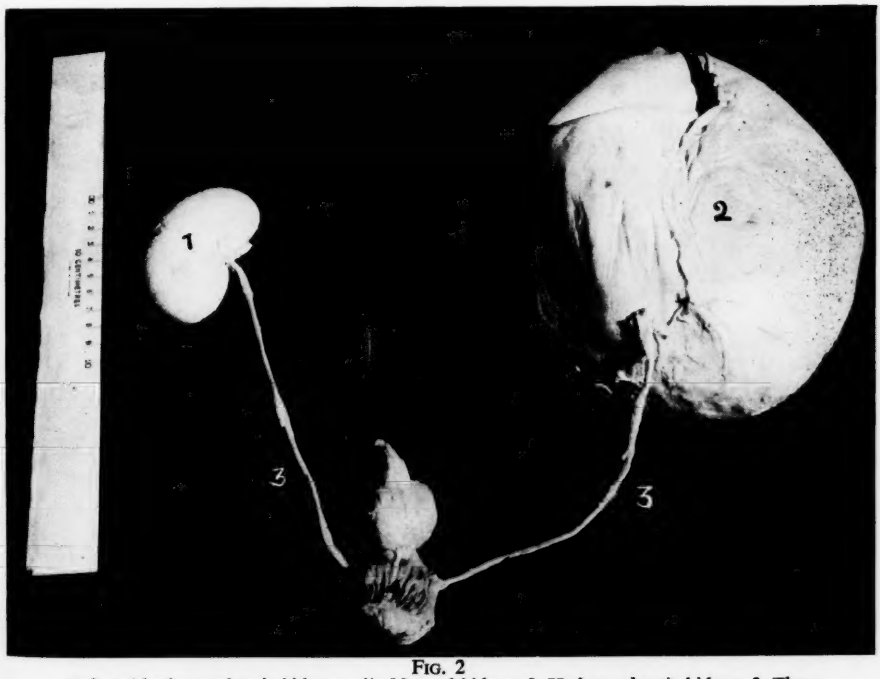

FIG. 2
Infected hydro-nephrotic kidney. (1. Normal kidney. 2. Hydronephrotic kidney. 3. The ureters and 4. Urinary bladder). (Dog. A.273/52).

(Article by Chandrasekharan, page 10)

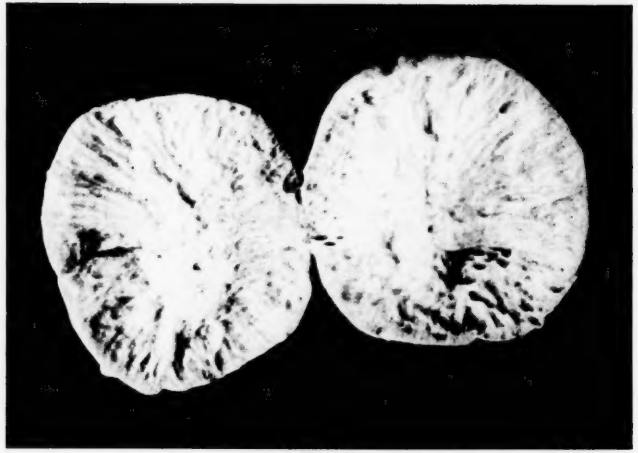

FIG. 3
Polycystic kidney, congenital—kid. Note spongy appearance of the kidney tissue. (Dog. Path. 276/54). 2/3rds natural size.

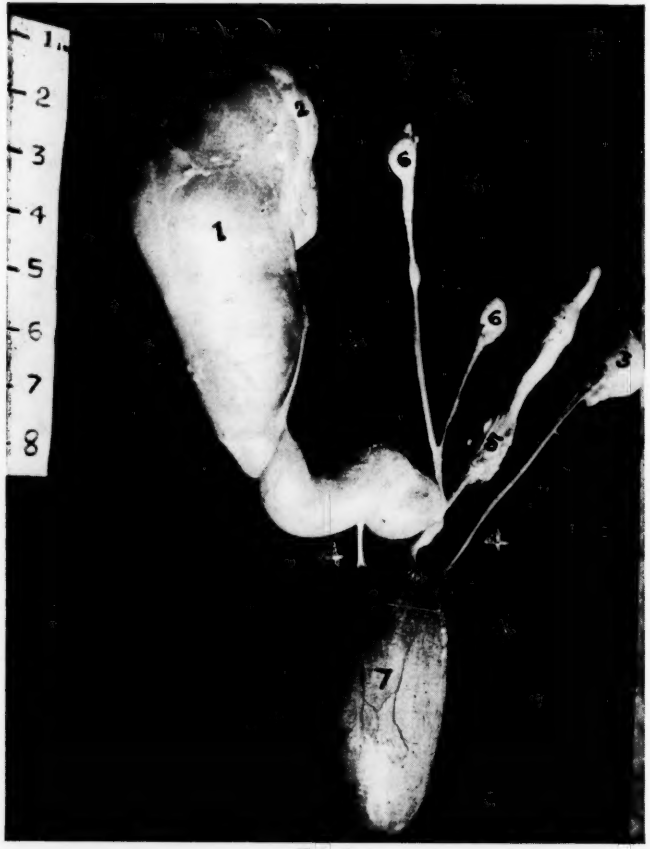

FIG. 4
Large solitary cyst in hydro-nephrotic kidney with other uro-genital organs.
1. The solitary cyst. 2. Right kidney showing severe hydronephrosis. 3. Left kidney showing mild degree of hydronephrosis. 4. Ureters. 5. Penis.

(Article by Chandrasekharan, page 10)

II. Infected Hydronephrosis—Dog

In dogs hydronephrosis has been met with occasionally in the course of routine autopsy work. All the cases were of the non-infected type and Damodaran (1950) working in this department has reported on them. The causes in these cases were calcular obstruction, metastatic tumour (lympho-sarcoma) of the renal pelvis and congenital stricture of the ureter. Since then we have also encountered advanced types of congenital unilateral hydro-nephrosis in a cat and a buffalo calf. The condition in dogs cannot be considered rare, but the case here described is worthy of report because of the enormous size attained by the affected kidney, the infection of the fluid and the difficulty attending the diagnosis of such patients when alive.

Case history: The subject was an Alsatian dog (male) about 1½ years admitted to the College hospital with the history that it had not been feeding well for one week. There was distention of the abdomen and ascites was suspected. The fluid which was yielded on paracentesis was chocolate brown in colour rich in pus cells and with occasional gram positive cocci. The animal, however, died and an examination post-mortem was conducted an hour later.

Autopsy (A.273/52): The carcase was fair in condition. The peritoneal cavity contained about 500 cc. of fairly thick chocolate brown turbid fluid. Evidently this fluid had escaped into the peritoneal cavity, due to a puncture made into the affected kidney, when paracentesis was performed. The right kidney was enlarged to nearly the size of a football (Fig. 3) causing displacement of the liver anteriorly and of the intestinal loops to the left of the median line. About 2 inches of the jejunum was adherent to the left lateral aspect of the kidney.

While the left kidney was normal in size and weighed 80 gms., the right measured 59×53 cms. and weighed 3.75 k.gms. On section about 3 litres of chocolate brown thick fluid escaped and practically no renal tissue could be seen. The capsule was thickened and slightly raised ossified trabeculæ could be seen internally. The ureter of the side was normal in thickness but on passing a blunt probe it was found to end more or less blindly at the pelvis and fuse with the connective tissue present at the area. Microscopical examination of the purulent chocolate fluid revealed numerous pus cells, crenated red blood corpuscles and gram positive cocci. It was alkaline in reaction and had an albumin content of 0.6 per cent. Cultural examination yielded Staphylococcus albus.

Comment: Hydronephrosis has been noticed occasionally by us in the past; but the cases were of the non-infected type containing clear fluid. Hydronephrosis of the type and extent described here is rare and to our knowledge, does not appear to have been reported in veterinary literature. The condition could be designated as " giant " hydronephrosis in accordance with the custom in human medicine wherein usually such a term is applied when the hydronephrolic fluid exceeds one litre. Considering the size of the animal which did not weigh more than 35 kgms., the amount of fluid present in the kidney was enormous. Obviously this had caused distention of the abdomen and the clinician, for the first time confronted with the condition, might diagnose the case as one of ascites. Since the affection in this

FIG. 4
Large solitary cyst in hydro-nephrotic kidney with other uro-genital organs. 1. The solitary cyst. 2. Right kidney showing severe hydronephrosis. 3. Left kidney showing mild degree of hydronephrosis. 4. Ureters. 5. Penis.

case was unilateral, no definite signs referable to the kidney were manifested. The vague signs reported by the owner led one to suspect a chronic affection of gastro-intestinal tract or liver.

The severe stricture or possibly complete occlusion at the uretero-pelvis region in this case is considered congenital on account of the young age of the subject and lack of inflammatory reaction at the site. In the human subject Bell (*loc. cit*) found that among 88 cases of hydronephrosis seen by him 18 were due to uretero-pelvic stricture. He believed that the majority of these were of congenital origin. In human subjects a large number of cases were of the non-infected type, whereas the case described here was of the infected type (Staphylococcal) both on gross and bacteriological examination.

III. Congenital Polycystic (Bilateral) Kidney—Kid

Though there are occasional references to polycystic disease of the kidney in animals, the condition is rare both in animals and man. Bell recorded 64 clinical forms among 32,360 autopsies performed by him. He found nearly 30 per cent of his cases in infants many of them being still-born. The rest of the cases were detected at later ages evidently due to slow development of the cyst; or it is possible that cysts might remain latent for years and then begin to increase in size. Usually in cases where the condition is detected late in life, the kidneys attain a fairly large size. They are easily palpable and invariably signs referable to renal insufficiency are present. In the infant type, besides the polycystic condition of the kidneys, congenital defects of other organs such as liver, pancreas, etc. are often seen.

The polycystic kidney described here was received from a veterinary institution for examination.

Case Report: A kid less than three days old was presented for treatment with a history that the abdomen was bloated. The animal died on the same day and on examination post-mortem the kidneys were found to be enlarged. One kidney was sent to the laboratory for examination and report.

Gross appearance: The specimen (*Path.* 275/54) was fixed in 10 per cent formalin. It measured 8×6 cms. On sagittal section innumerable small cysts with practically no renal tissue in between them were seen. The appearance was that of a loose spongy tissue (Fig. 4). No separation into cortex and medulla was possible and no calyces were seen. Considering the age of the animal, the kidney appeared to be much enlarged. The amount of fluid present in the cyst was very small, but by pressure on the renal tissue after section, a quantity of clear fluid oozed out.

Microscopic Examination: Sections made from different portions of the kidney showed the typical features of infant polycystic kidney. The cystic spaces seen macroscopically were evidently dilated portions of tubules or capsular spaces. They were lined by cubical, columnar or flattened cells. Though usually single-cell deep, occasionally at places the lining cells were seen in many layers in a heaped up fashion. The connective tissue stroma in between these spaces was considerable. There was a great reduction in the number of glomeruli and the few present were scattered in the cortical portion. They also appeared to be smaller in size. Neither glomeruli nor the vessels in the kidney showed any evidence of sclerosis.

Comment: Much work on the pathogenesis of the condition has been done in

the human subject. There is no doubt as to its congenital origin and very often along with this lesion, other congenital defects such as cystic liver or pancreas have been noticed in infants. A hereditary tendency has been noticed also.

Regarding the genesis of polycystic kidney many theories have been put forward from time to time and for the proper understanding of these some knowledge on the pre-natal development of the organ is essential. It may be recalled that in the normal development of the kidney, the primitive ureter from the Wolffian duct grows cranially to meet the metanephric blastoma. No tubules could develop if this meeting does not take place. The primitive ureter develops distally to form calices and from these the collecting tubules are formed by repeated division and subdivision. Each generation of the collecting tubules comes into contact with the blastoma which forms a solid cap over its end. A lumen is then formed in the latter which communicates with that in the collecting tubules. In this manner an original nephron is formed, the glomerulus being formed at the opposite end of the convoluted tubule. Normally the first few generations of convoluted tubules are not permanent structures. They become detached from the collecting tubules but persist for some time as cysts and then atrophy. If they persist, it is suggested that they form the innumerable cysts present in the polycystic kidney. Work of Kampmeier (1923) who studied the reconstruction of the cyst, proved this kind of origin.

Another concept originally suggested by Ribbert (1900) was that there was failure of union of newly formed collecting and convoluted tubules and that the former then developed into cyst. The basic objection to this hypothesis is that unless there was the contact of the ureteral portion with metanephric blastoma, no tubules are formed. Hence the non-union of the two portions with cystic dilatation of the collecting tubules is not possible.

Yet another theory put forward by Sleamler (1921) suggested that polycystic kidney represented a neoplastic process, possibly a cystadenoma.

The majority of workers are in favour of the first mentioned hypothesis, proved correct through extensive reconstruction studies on the diseased kidney. The cysts may in fact may be any portion of the tubule or capsular space and many of them are of the retention type. They undergo progressive enlargement and as a rule they do not open into the renal calices.

In the case described here, it is clear that the lesion is of congenital origin and that the genesis of the condition is in no way different from that in the human beings. Evidently the death of the kid within three days of birth was due to extreme renal failure caused by the condition.

IV. Solitary (sub-capsular) Cyst of the Kidney with Hydronephrosis—Dog

Besides the poly-cystic kidney, large sized solitary cysts are occasionally described in human beings. The condition is very rare and Bell (*loc. cit*) in a survey found only 7 examples of large solitary cyst amongst the 32,360 autopsies performed. Depending upon the size of the cyst, it may cause varying degrees of compression of the kidney and the adjacent viscera. The cysts may contain serous or hæmorrhagic fluid and are often uni-locular with a thick fibrous wall. Usually it is lined internally by a single layer of cubical epithelium. The relationship of the cyst to the

renal tissue is important. It may or may not communicate with the pelvis and might be included with renal tissue. In other cases it may be outside the renal capsule and communicate with the pelvis or it may be sub-capsular with no communication with the pelvis. The first two are the more frequent forms and the other two are of the rarer types.

To our knowledge large solitary cysts of the kidney have not been reported in veterinary literature. Because of this, the case reported here is considered interesting.

Case history: A cross bred male dog, aged 3 years, poor in condition with history (on 9.12.54) that it has been " unwell " for a month and not able to see. In spite of treatment the dog died on 14.12.54.

Autopsy findings : On opening the carcase a large cyst was seen on the lateral aspect of the right kidney and this extended for a considerable distance towards the pelvic region. The intestinal loops were displaced to the left side and the liver was compressed and displaced anteriorly. About 6 in. of the cranial part of the small intestine was adherent to the medial surface of the cyst. The cyst measured 14½ in. in length and the maximum circumference of 17 in. was at the unattached portion of it, which was at a distance of about 2 in. from the posterior pole of the kidney. The cyst was roughly oval at its attached portion, whereas at its free portion it assumed a tubular shape, the diameter of which progressively diminished towards the pelvis. At the level of the urinary bladder the cyst measured about 6 in. in circumference. It was sub-capsular in position and showed no communication with the renal pelvis. It had a fibrous wall and the internal membrane was smooth. It was unilocular and contained about 2 litres of clear straw-coloured fluid with acid reaction.

The affected kidney also showed extensive hydronephrosis, the fluid present being about 500 cc. Only a small amount of renal tissue was present at its anterior pole. The ureter of the kidney was dilated considerably (about 2 in. in diameter). It was thin walled and adherent to the internal aspect of the cyst all along its course, excepting for its final 2 in. This portion of the ureter had a thick wall and was of normal size. The hydronephrotic fluid was slightly turbid, with specific gravity of 1020 and it contained traces of bile pigments.

The left kidney showed some amount of distension of the pelvis and anterior portion of the ureter, but the kidney tissue was unaffected.

The testicles were atrophic and intra-abdominal, and placed lateral to the respective kidney.

Comment: Large solitary cysts of the kidney are considered rare in man and undoubtedly so in animals. The signs shown by the patients are often indefinite and it is difficult to diagnose in life. Those that occupy a retroperitoneal position cause gastro-intestinal disturbances due to compression of the adjacent viscera. The chief sign in this case was that the animal had not been feeding well. This may have been due to the slowly developing cyst. The advanced hydronephrosis was due to compression of the ureter by the developing cyst and the adherence of greater part of the former on to the latter.

Regarding the pathogenesis of solitary cysts Bell (*loc. cit*) states that nothing definite is known and they are presumably of congenital origin. Their infrequent

detection in early life, he believes, may only be due to slow development. In the case reported here the cyst began to show clinical evidence about the age of 1½ years only. Its presence along with another congenital anomaly, viz., cryptorchid testicles makes one tend to support the idea of a congenital origin.

Summary

A few uncommon diseases of the kidney in animals are described. They were encountered in the course of diagnostic work in the Department of Pathology of this College over the past six years. The cases include two of lipomatosis ox; infected hydronephrosis, dog; congenital bilateral polycystic kidney, kid; and large solitary (sub-capsular) cyst with hydro-nephrosis, dog. The lesions were not diagnosed during life owing to the rarity of the diseases and the indefinite signs presented. In each case comments are made in the ætiology and pathogenesis and the lesions were compared with the corresponding conditions in human beings.

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REFERENCES

Bell, E. T. (1947): Renal Diseases, Lea & Febiger, Philadelphia.
Damodaran, S. (1950): *Ind. Vet. J., 27,* 192.
Edgson, F. A. (1952): *Vet. Rec., 64,* 449.
Hamn, F. C. and De Veer, J. A. (1939): *J. Urol., 41,* 850.
Kampmeier, O. F. (1923): *Sur. Gynec. Obst., 36,* 208.
Kuttzman, A. A. (1931): *Surg. Gynec. Obst., 52,* 690.
Priestley, J. B. (1938): *J. Urol., 40,* 269.
Ribbert, H. (1900): *Verhandl. d. deutsch. path. Gesellsch., 2,* 187. Cited by Bell, E. T. Renal Diseases, 1947.
Sleamler, M. (1921): *Beitz. Z. Path. Anat., 68,* 22. Cited by Ruffle, B. M. and Jackson, R. B., 1955. Nat. Cancer Int., *15,* 1183.
Young, H. H. (1932): *Trans. Am. Genito-urin-surgeon, 25,* 105. Cited by Bell, E. T. Renal Diseases, 1947.

A CAUSE OF MALFORMATION OF THE LIMBS OF SHETLAND PONIES WITH A NOTE ON ITS PHYLOGENIC SIGNIFICANCE

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DEFECTS of the hocks are not uncommon in horses and ponies. One type of malformation appears in some strains of Shetland ponies and in view of the great popularity of Shetland ponies and the impetus their breeding has recently received, an opportunity to examine the condition was welcomed.

Material

Three pedigree Shetland pony foals, two colts and one filly, each aged four months, were brought in for examination. Each foal had been noted to have an awkward gait in one or both hind legs. The foals were born on open hill country and had run with their mothers. They were in good condition and although it had not been possible to keep them under close observation from birth, it was reasonably certain that the onset of their "lameness" had not been sudden. The lameness was becoming more noticeable as time went on. When the foals were standing still and viewed from the rear, the hind limbs appeared to be normal down to the hock. The upper part of the hocks was prominent on the inner aspect where they were close together and superficial examination and palpation revealed no other obvious abnormality of the limbs. They were not "cowhocks" since there was no appearance of outward rotation. Below the hocks the limbs splayed outward. In two of the foals both hind limbs appeared to be affected, and in the other only the near-side limb. When walking the action of the hind legs was abnormal, the lower part of the limb swinging outward markedly when being carried forward. This feature became more pronounced with faster movement and the animals were most reluctant to trot. It was the view of several people who had experience of Shetland ponies that this condition was not rare and the prognosis was that as the foals grew older the condition would not improve. X-ray pictures of the affected limbs were made and then the animals were destroyed.

X-ray Findings

In the X-rays taken in the living animal the only remarkable thing was a shadow lying along the lateral aspect of the tibia, in a position similar to that occupied by the fibula in the dog or pig. The epiphyseal line at the distal end of the tibia was distinct medially, but laterally less so. To confirm these observations, when the animals had been killed the skin and much of the muscles were removed and further X-ray pictures were made of the bones.

Dissection

Nothing unusual was noted in the dissection of the hip-joint and thigh. The stifle-joint seemed normal. The leg and the hock-joint however were distinctly abnormal. As was deduced from the shadow lying along the lateral border of the tibia

in the X-ray pictures, that the fibula had a shaft which connected a head or proximal extremity with a distal extremity or lateral malleolus. The proximal extremity articulated with the lower part of the lateral border of the lateral condyle of the tibia by a syndesmosis, while the distal extremity was near the distal extremity of the tibia. The shaft curved towards the tibia and had a roughened area near its middle where it was in contact with that bone; above this roughened area there was a wide interosseous space between tibia and fibula, while below it the two shafts were closely attached to each other by fibrous tissue. This fibrous tissue became progressively better defined towards the distal extremities of the shafts forming a distinct transverse ligament. Continuous with this transverse ligament and uniting the distal extremity of the fibula with that of the tibia were two ligaments, one anterior and one posterior. These were comparable with the anterior and posterior lateral malleolar ligaments of some other animals.

The musculature of the limb was well developed. Only two differences from the normal were noticed, in each case influenced by the presence of a fully developed shaft of the fibula. These differences were, that the peronæus longus muscle (lateral digital extensor) took origin from a considerable portion of the shaft of the fibula but not from the tibia and that the flexor hallucis longus also had an extensive origin from the shaft of the fibula in addition to its usual origin from the posterior aspect of the tibia.

The slope of the articular surface formed by the distal ends of tibia and fibula was distinctly oblique, sloping upward from medial to lateral. Both hind limbs in each of the foals showed the same features, differing only slightly in degree.

Discussion

The presence of a full-length shaft of the fibula in a horse or pony is an example of " atavism " since it represents a phase in the evolution of the equidæ which ended some millions of years ago. In itself the presence of this shaft should not interfere with conformation or locomotion. In modern horses the shaft of the fibula is reduced, sometimes to a mere spicule attached to its proximal extremity, but sometimes several inches long, slender, and perhaps continued to the distal extremity by a fibrous cord. In Shetland ponies this proximal part of the fibula may be fused with the lateral condyle of the tibia as in the ox, not forming a syndesmosis with it as in most horses and ponies. Usually the distal end of the fibula is separate from its shaft but it is united with the distal end of the tibia where it forms the lateral malleolus. There is one continuous epiphyseal line for this distal end of the fused tibia and fibula. In the present cases these distal extremities were separate from each other and each had its own epiphyseal line.

Where a full length shaft and a distal extremity are present in the fibula and this distal extremity is separate from the tibia, the articular surface thus provided for the proximal row of tarsals will depend for its alignment and weight-bearing capacity upon the two bones maintaining their relative positions. To ensure this, equal growth in tibia and fibula is necessary. Should the tibia lengthen more rapidly than the fibula, obliquity or distortion of the articular surface offered by the distal ends of tibia and fibula would occur, as in the present specimens. This in turn could cause the limb below the tibio-tarsal and fibulo-tarsal articulation to be deflected outward.

It was noted during the dissection of the malformed limbs that the tibia articulated only with the tibial tarsal, while the fibula articulated with both tibial and fibular tarsals. Such a condition resembles that which existed in the Miocene horses and is one in which the fibula is an important weight-carrying bone of the leg. This therefore had important bearing on the progress of the defective condition in the hock-joints of these Shetland pony foals, in that the fibula was needed as a fully functional primitive weight-carrying outer bone of the leg. Since the mere presence of a shaft of the fibula, as has already been suggested, should not cause inconvenience or interference in function, it can be understood that, although the presence of a complete shaft and of a distal end of the fibula separate from the tibia represents an atavism, the disfunction and malformation of the hocks resulted from defects in the atavistic structure rather than from the presence of the structure itself. An examination of the fibula indicated that the defects could have been partly mechanical and partly physiological. The shaft of the fibula was greatly curved in its length, attached closely to the tibia about the middle but separated from it by a wide interosseous space above and by a smaller one below. Mechanically this curved shaft would be less effective in supporting weight than a straighter one would be. In addition to its curvature, the shaft was relatively slender and out of proportion to the large distal extremity with its large articular areas for both tibial and fibular tarsal bones. The physiological defect appeared to be in the distal epiphyseal line of both tibia and fibula, for in the tibia the medial part of the line was open while towards the lateral aspect it was either closed or had not existed. In the fibula the distal epiphyseal line was almost closed. From this observation it was evident that growth at the distal extremities would be unequal and that the slope of the articular surface would become progressively greater.

The causal factors are not obvious. The condition appears to be hereditary since it recurs in certain families. It may miss a generation or two and then reappear and foals suffering from the condition may be produced by parents which are apparently free from it but which belong to strains where the condition has been noted previously. It is possible that the abnormality is related to dwarfing which is aggravated by selection of short-legged animals for breeding purposes. In this context it is worth noting that the Shetland pony does not conform in size and proportions to any prototype horse and that its fossil remains occur only in association with modern man. Its nearest relatives are probably those mountain ponies which formerly were common in Britain and in the Western Isles, notably Barra. These are now most characteristically represented by the Exmoor pony which has retained much of the original form and colour pattern. The fossils of such ponies are common enough in association with palæolithic man and indicate fairly accurately that the height of this race or subspecies of horse was 12.2 hands or 50 in. or 128 cms. and that it existed in Britain as an indigenous animal. The Shetland ponies appear to be derived from such ponies primarily, but have since been modified to their present form.

It is a matter of importance that it should be clearly understood that there is no evidence that the dwarfing of the Shetland and other small races of horses is caused in the first instance by environmental conditions. Although the small horses

PLATE I

FIGURE 1
Shaft and distal extremity of tibia in the leg of a normal young Shetland Pony

FIGURE 2
The X-ray appearance of the leg of one of the Foals

FIGURE 3
One of the abnormal limbs partially dissected

FIGURE 4
The skeleton of an abnormal leg and foot from the rear

(Article by J. G. Speed, page 18)

PLATE II

FIGURE 5
A dissection to show the "Malleolar" ligaments

(*Article by J. G. Speed, page 18*)

of the Celtic peoples, or of the " La Tenee " culture, are the ones best known to history, it can be demonstrated at any time and in any climate or environment that introduction of a different race into any native horse population leads to changes in it. In the absence of control by culling and careful selection these changes for a period commonly result in degeneration in conformation and size, even if at first there may have been some evidence of " improvement." Provided the degeneration does not go beyond the race's powers of survival an ultimate return to something resembling the original native race can be expected to take place. If however, environmental hardships aided by selection by man should act against the natural regeneration process, an arrested form of regeneration may be preserved. This appears to be the case in the Shetland pony. It is, probably, a representative of the native mountain ponies but altered from its original form first by being outcrossed and selectively bred, presumably for chariot use by Celtic peoples, and then kept metaphorically—or actually—in cold storage on an island. That it has developed a reasonably stable genetical pattern over its two or three thousand years of existence is sure, but there is always the possibility of an attempt at regeneration or a return to the 12.2 hands height of the native mountain pony, its chief ancestor. The danger exists that in selecting breeding stock of very small size actual dwarfs may be chosen. Of the bones of the limbs which may show dwarf forms by reduction in length of the shaft, the likeliest in horses are the humerus, femur, radius and tibia. Of the bones of the head, it is the mandible, incisive bones (premaxillæ), the nasal bones and the maxillæ which most commonly are disproportionately shortened. The conclusion is that in artificial selection for small size, animals which suffered from such forms of degeneration as " atavism " or disproportion in the form of dwarfishness, may be chosen for breeding.

Fore-limbs

An interesting commentary on the condition of the hind limbs was provided by that of the fore limbs. Here there was present a shaft of the ulna, complete and joined by a long epiphyseal cartilage to its distal extremity. This distal extremity was fused with the distal extremity of the radius and provided only part of the articular surface for the ulnar carpal, the rest being provided by the radius. Probably as a result of the fusion of the distal end of the ulna with that of the radius, the articular surface provided for the proximal row of carpal bones by the combined radius and ulna had remained level and horizontal in spite of any weakness, slenderness or other defects of the shaft of the ulna. The epiphyseal junction with the shaft was situated one centimetre higher in the ulna than in the radius and in a dried specimen there was a gap between distal extremity and shaft in the ulna of half a centimetre. This gap was occupied by the epiphyseal cartilage in a fresh specimen.

Examples have been seen of deformities in the fore limbs of Shetland ponies which could possibly be ascribed to the presence of full length ulnae, but these had not been available for anatomical study in the preparation of this paper.

Conclusions

The conclusions drawn from the three cases which are at present being described are:—

1. that fibulæ and ulnæ were developed to a degree which is abnormal in modern horses, the abnormalities in the former causing signs of lameness: the presence of the latter had no obvious ill effect.
2. that the presence of these bones in a primitively advanced degree of development may be considered to be evidence of either atavism or dwarfing.
3. that the existence of the condition suggests that a study of the proportions of the various long bones of the limbs and their ratios to each other in normal Shetland ponies would be of value in determining the proper conformation these animals should possess and to exclude the danger of breeding from dwarfs.

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A CASE OF EOSINOPHILIC MYOSITIS IN A DOG

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Introduction

In view of the increasing interest focused on Eosinophilic Myositis of dogs in this country following the publications of Harding and Owen (1955), Whitney (1955) and Clare (1956), we felt that the details of a case of this disease confirmed at post-mortem examination would be of interest. During the period from 1st August, 1951, to 31st July, 1955, one thousand six hundred and fifty-two dogs were subjected to port-mortem examination at the R.(D).S.V.S., one hundred and thirty-one of which were Alsatians. The case described below was the only example of Eosinophilic Myositis discovered in these carcases; the majority of these dogs had lived in the city of Edinburgh and its environs.

Subject

A pedigree sable Alsatian bitch, born in Edinburgh on 29th June, 1947. The animal was obtained by its owner on the 13th of October, 1947, and since that date had lived in a small mining village near Edinburgh.

Past History

The animal was fed a normal mixed diet though the protein was provided by fish rather than by meat; it was given a large size Bob Martin condition powder at regular intervals. Since the dog was kept as a pet, it only went out with its owner who took it for one walk each day and occasionally for longer walks. Oestrus cycles were normal and regular every February and August. The bitch had never been mated and she had not been vaccinated against distemper while in the owner's possession. There was no history of any serious disease; special enquiries were made with negative results about distemper (fits, chorea, etc.), lung trouble, tonsillitis, heart disease, lameness and intestinal disorders. When a puppy, it developed a dry eczema in the axilla which responded readily to Calamine lotion. According to the owner, the bitch was of " sweet disposition " and there was no history of blows around the head or neck.

In May 1950 when about three years old, the muscles of the head became hard and stiff and although the animal wanted to eat, it was unable to do so. There was no protrusion of the eyeballs. After fourteen days, the dog recovered spontaneously. A similar but more severe attack with protrusion of the eyeballs occurred in August 1951. This attack resolved spontaneously after about fourteen days.

Immediate History

On 12th May, 1952, the head muscles again showed enlargement and eating became difficult although the bitch did manage to eat some food. She was first examined by one of us (W. MacLennan) on 13th May, when she was found to be in good condition, bright and intelligent. The temperature was 103.6° F. The pulse rate and respirations were normal. Auscultation of the chest and abdominal palpation revealed no abnormality. The temporal and, to a smaller extent, the masseter muscles were symmetrically enlarged, felt doughy but did not " pit " on pressure. Unlike the cases reported by Ullrich (1950) these muscles were not hot or painful. The submaxillary, retropharyngeal and parotid lymph nodes were enlarged, firm in consistency but no heat or pain could be appreciated. No other superficial lymph nodes were palpable. The eyeballs protruded slightly. The jaws could be opened wide without exerting much force, but the dog resented this. The soft palate was enlarged, œdematous, and congested. The tonsils could not be examined as the dog objected to the tongue being depressed. The tongue and teeth appeared normal. The dog could open its jaws slightly and seized its food easily but showed difficulty in swallowing solid food though it swallowed fluids normally when drinking. Defæcation and urination were normal; the polyuria noted by Wirth (1941) and Clare (1956) was not observed.

The dog was treated by daily intramuscular injections of 300,000 i.u. Procaine Penicillin for three days during which time the temperature only dropped to 102.8° F. This is in accord with the observations of Winter and Stephenson (1952). By the 16th May the enlargement of the muscles of the head had become much more marked although the dog was still bright, was eating well and had not lost condition. The eyeballs showed more protrusion, were very painful and there was slight hæmorrhage from the exposed congested conjunctiva. The membrana nictitans was protruded across the eyeball. On the basis of the blood findings described below, a

diagnosis of Eosinophilic Myositis was made and the bitch was destroyed by intravenous injection of Pentothal sodium.

Blood Findings

Erythrocyte Picture: Basal Sedimentation Rate—2 mm. in 1 hour; Packed Cell Volume—42 per cent (½ hour at 3,500 r.p.m.); Hæmoglobin—13.6 gm./100 ml.; Total R.B.C. Count—4.5 million per cm.; Mean Corpuscular Volume—93.3 c/u., Mean Corpuscular Hæmoglobin Concentration—32.4 per cent; 10 normoblasts were seen when the white differential count was being made. The R.B.C.s showed slight anisocytosis and a moderate degree of polychromasia. These findings indicate a slight macrocytic, normochromic anæmia with regeneration and are similar to those mentioned by Kuscher (1940) and Whitney (1955).

Leucocyte Picture: Total W.B.C. Count—18.6 thousand per cmm. The differential count showed: Non-lobulated Neutrophils—4.5 per cent (837); Lobulated Neutrophils—62.5 per cent (11,625); Eosinophils—13 per cent (2,418); Lymphocytes —16 per cent (2,976); Monocytes—2.5 per cent (465). These findings indicate a slight leucocytosis which is mainly due to eosinophils. All the eosinophils had one or two lobes to their nuclei, i.e. they were adult forms as in Wirth's case (1941) and Harding and Owen's cases (1956)—with the exception of their case 2 at the third attack.

Post-Mortem Findings

The carcase was well developed and in a moderately good state of nutrition. The messeter, temporal, pterygoid and digastric (synn. occipito-mandibularis) muscles showed bilaterally symmetrical enlargement. On section they were slightly œdematous with widespread pale mottling and areas of hæmorrhage. Minute white to yellow elongated foci were scattered throughout these muscles (Fig. 1). The eyes appeared normal except for a slight conjunctivitis most noticeably involving the third eyelid; protrusion of the eyeballs was presumably due to the enlargement of the muscles detailed above. The parotid lymph nodes measured 2 cm. diameter; the three submaxillary lymph nodes each measured $2.5 \times 2.5 \times 1$ cm.; the retropharyngeal lymph nodes measured $7 \times 3 \times 2$ cm.; the enlargement of these lymph nodes was due to follicular hyperplasia of the cortex with congestion and œdema of the medulla. The tonsils were normal.

The nasal passages, larynx, trachea and main bronchi showed no obnormality. There was slight anthracosis of the lungs with 0.25 cm. diameter greyish plaques of pleural fibrosis on the dorsal surfaces of all lobes, particularly on the diaphragmatic lobes. The bronchial lymph nodes were normal. The pleural surface of the diaphragm showed two calcified elongated yellowish lesions slightly larger than those seen in the head muscles.

The tongue showed a 0.25 cm. elongated calcified lesion on the left side of the dorsal surface. The remainder of the alimentary tract was normal apart from occasional 0.25 cm. diameter calcified yellow plaques visible through the serosal surface of the small intestine. The mesenteric and portal lymph nodes were normal.

The liver was normal. The spleen was slightly engorged as in the cases of Harding and Owen (1956) and Clare (1956). The heart showed marked left ventricular hypertrophy and under the endocardium of the papillary muscle on the

outer wall of the left ventricle there was a group of five 0.25 cm. calcified yellow lesions. Small nodular hyperplastic lesions were observed in the adrenal cortex.

The capsule of the kidneys stripped with difficulty, revealing shallow pits on the surface and multiple pin-point yellow calcified foci, some of which remained on the inner surface of the capsule. The cut surface of the kidney was normal. The ovaries were small, fibrous and showed a few regressing corpora lutea. The uterus, vagina and bladder were normal. The brain and cranial cavity showed no abnormalities.

Bacteriological Examination

Cultures prepared from the retropharyngeal lymph node were sterile. Cultural examination of the masseter muscle resulted in the isolation of a gram negative bacillus which, from its biochemical and biological reactions, appeared to belong to the *Ozæna-rhinoscleroma* group of organisms and is similar to Perez's bacillus.

Helminthological Examination

Compressorium preparations of the diaphragm lesions and head muscles failed to reveal any helminth larvæ. Examination of the gut contents and fæces failed to reveal any helminths or eggs.

Histological Examination

Portions of liver, kidney, heart and head muscles were fixed in 10 per cent formal saline, processed by frozen section technique and stained with Sudan IV. All tissues were fixed in 10 per cent formol saline and in Du Bosque fixative (picric-acid-alcohol-formol), processed paraffin wax and stained Anderson's hæmatoxylin and eosin. Prussian Blue reaction, iron alum hæmatoxylin after the method of Heidenhain, Gordon and Sweet's method for reticulin, Ziehl Neelson's method, Von Kossa's method for calcium and Kull's stain were used on appropriate sections.

The masseter, temporal, pterygoid and digastric muscles all show similar changes. The perimysium is infiltrated by lobulated eosinophils and a few macrophages. The arterioles are normal; the venules are normal but contain a large number of eosinophils and lymphocytes in their lumina; the nerves are normal. There is no increase in collagen fibres or in the reticulin network in sections stained by Gordon and Sweet's method. Occasional groups of adult fat cells are present.

The endomysium shows a similar diffuse infiltration of cells but there are an equal number of plasma cells, lymphocytes, macrophages and eosinophils. There is no increase in the number of reticulin fibres (Gordon and Sweet's method). The muscle fibres in general show no abnormality; their striations are not distinct in sections stained H. & E. but can be shown clearly with Gordon and Sweet's method and Heidenhain's hæmatoxylin staining (Fig. 5a). No fat droplets are visible in the sarcoplasm of the muscle fibres.

There are focal lesions in these muscle bundles often near the perimysium but sometimes deep in the muscle bundles. These foci are composed of very numerous lobulated eosinophils (well demonstrated by Kull's stain) often situated in the centre of the focus, many macrophages and groups of plasma cells and lymphocytes. Occasional multinucleate giant cells are visible (Fig. 2) which are much smaller

than the degenerating muscle bundles described below. Very few reticulin fibres run into these cellular foci (Fig. 3). In some of the larger foci there is central necrosis of a coagulative type with "ghost outlines" of inflammatory cells but no trace of "ghost outlines" of muscle fibres. Some of these foci show hæmorrhage, presumably from capillaries, and some œdema. Only a very few macrophages containing hæmosiderin are visible in sections stained Prussian Blue reaction. The muscle fibres running into these cellular foci in sections stained H. & E. show enlargement of the sarcolemmal nuclei, which form groups and chains, and tend to become central (Fig. 2); the sarcoplasm becomes light pink and vacuolated centrally with a dark red condensed periphery (Fig. 4). A few atrophied fibres represented by rows of pyknotic sarcolemmal nuclei are seen. These various muscle degenerations have been described by Harding and Owen (1956). With Gordon and Sweet's method and Heidenhain's hæmatoxylin, these degenerating fibres show loss of transverse striation and the whole fibre stains more intensely; then the sarcoplasm breaks-up and loses its staining affinity, leaving irregular coarse transverse bands (Fig. 5b).

It is of interest to note that there is no evidence of regeneration of the relatively healthy muscle fibres either by budding or by proliferation of sarcolemmal nuclei. This may be because the present lesion is only of four days' clinical duration. The picture is intermediate in severity between Kuscher's (1940) case and Winter and Stevenson's (1952) case or Whitney's (1955) cases II and III and is very much more active than Erichsen's (1952) case. No light is thrown by these sections on the problem as to why there is no fibrosis following the previous attacks. As in the previous reports in the literature, no Trichinella larvæ or Sarcosporidia are visible. Toxoplasmal pseudocysts were not seen.

The eyes show no abnormality. The lymph nodes of the head show follicular hyperplasia; the sinuses of the nodes contain some free R.B.C.s, a few eosinophils and moderately numerous macrophages full of hæmosiderin (Prussian Blue reaction). The lungs are anthracotic, the smaller bronchioles show a mild low-grade bronchiolitis and the lesions noted macroscopically on the pleura are focal areas of collagen formation. The mucosa of the small intestine shows superficial necrosis and desquamation, the deeper layers of the mucosa being infiltrated by plasma cells and lymphocytes. The lesions noted macroscopically lie in the circular muscle coat and consist of a mass of calcified debris (Von Kossa's method) surrounded by a fibrous capsule; there is no transition between the inner zone of plump fibroblasts and the calcified tissue. The liver is congested, the Kupffer cells containing hæmosiderin (Prussian Blue reaction) and there is slight centrilobular infiltration by plasma cells and lymphocytes as reported by Harding and Owen (1956). The spleen is slightly engorged, the red pulp containing fairly numerous macrophages full of hæmosiderin (Prussian Blue reaction). The heart muscle shows no abnormality apart from the lesions noted macroscopically which consisted of a mass of dense collagen fibres with few nuclei, surrounding irregular areas of calcified debris. The cells of the cortical tubules of the kidney show cloudy swelling; the lesions noted macroscopically are interstitial foci of plasma cells, lymphocytes and macrophages around a central area of necrosis containing a few coccobacillary bacterial forms (Ziehl Neelson's method).

The orbital muscles and the muscles of the stomach and small intestine are normal. The lesions in the small intestine, heart and kidneys show no toxoplasmal pseudocysts and in sections stained Zeihl Neelson's method, no acid-fast or other organisms are visible.

Discussion

In the period mentioned above, four cases of bilaterally symmetrical atrophy of the masseter and temporal muscles were observed and in these cases considerable force was needed to open the jaws (c.f. eosinophilic myositis). None of these cases had a history of a previous swelling of the muscles of the head region. Only one of these dogs showed an eosinophil leucocytosis shortly before death (Table 1). Since Wirth (1941), and Whitney (1955) have suggested that sub-clinical cases of eosinophilic myositis may occur in which the acute stage of swelling was not observed in the muscles, and since in Clare's (1956) case the percentage eosinophil count was normal until at least one year after the first onset of symptoms, a few details of these cases are worthy of note.

Case 1: A two-year-old male Whippet showed a progressive wasting disease of all muscles but especially those of the head for four months prior to destruction. Post-mortem examination revealed a tuberculous pleurisy and pneumonia with hæmatogenous spread to the liver. Biological and cultural examination of the strain of *Mycobacterium tuberculosis* isolated showed it to be of the bovine type. Histological examination of the masseter and temporal muscles revealed atrophy of the muscle bundles, fibrosis, slight hæmorrhage and a light diffuse infiltration by plasma cells, lymphocytes, macrophages and neutrophils. No acid-fast organisms could be detected in these muscle lesions.

Case 2: A seven-year-old male Welsh Corgi which had distemper when young and showed chorea for two years before destruction. The jaws were malformed in that the maxilla protruded one inch beyond the mandible. Histologically the brain showed a demyelinating encephalitis of the type seen in long-standing infections by the distemper complex of virus diseases. The masseter and temporal muscles showed atrophy and fibrosis but with no foci of hæmorrhage and no cellular reaction.

Case 3: A three-year-old male Welsh Corgi which had a history of periodic attacks of lameness with progressive wasting of the masseter and temporal muscles of eight months' duration. Post-mortem examination revealed a low-grade arthritis of all major joints, including the temporo-mandibular articulation. *Streptococcus viridans* was recovered in pure culture from these joints. The masseter and temporal muscles show atrophy and fibrosis but with no hæmorrhage and no cellular reaction.

Case 4 (mentioned by Harding and Owen, 1956): An eighteen-month-old female black and white Collie which liked to chase cattle and bite their tails. Three weeks before destruction, the animal suddenly developed difficulty in eating, the muscles of the head started to sink in and the eyes became sunken. The difficulty in opening the mouth became progressively more marked until the dog could not open its mouth and even after general anæsthesia, two people were needed to open the jaws. The only significant findings on post-mortem were bilaterally symmetrical atrophy of the masseter and temporal muscles and recent blood coagula in both frontal

sinuses. The bones of the skull showed no fractures. On histological examination, the masseter and temporal muscles show focal aggregations of macrophages and polymorphs with a few eosinophils and mast cells around foci of degenerating muscle bundles. This case is similar to those described by Robin and Brion (1934) and Whitney (1957) under the name of atrophic myositis.

TABLE 1
Total W.B.C. Count, Differential and Absolute Eosinophil Count of four cases of Dogs with Atrophy of the Head Muscles

Case Number	Leucocytes per cu.mm.	Eosinophils per cent	Eosinophils per cu.mm.
1 Day of death	13,600	4	544
2 Day of death	13,500	3	405
3 15 days before death	43,400	0	0
3 days before death	48,100	2	962
4 Day of death	12,000	10	1,200

The recovery of a member of the group of the *Ozæna-rhinoscleroma* organisms does not appear to have been significant as is shown by the following experiments. An adult male rabbit was inoculated with 1 cc. of the eosinophilic myositis muscle triturate in normal saline into the thigh muscle of one leg. No alteration was noted in the total or differential W.B.C. count of the rabbit over the following nineteen days. On the twenty-sixth day after inoculation the animal was slaughtered and although bacteriological examination of the thigh muscle resulted in the isolation of a similar bacterium to that recovered from the masseter muscle of the dog, no macroscopic or microscopic abnormality could be detected in these muscles or, indeed, in the whole animal. Two albino mice were inoculated with ½ cc. of broth culture of this organism into the thigh muscle on one side. No illness was noted in the following twenty days; the animals were therefore slaughtered and on post-mortem and histological examination, no significant abnormalities were detected in the thigh muscles.

The type of nodular hyperplasia of the adrenal cortex present in this case is commonly seen in dogs (Tamaschke, 1955) and should not be correlated with the response of eosinophilic myositis cases to Cortisone (Moon and Wood, 1950) and A.C.T.H. (Whitney, 1955; Clare, 1956; and Harding and Owen, 1956).

The significance of the lesions in the diaphragm, tongue, small intestine and heart is not understood but they may be the end result of the local cellular lesions noted by Harding and Owen (1956) in the tongue and other muscles. One could not, however, explain the lesions in the kidney on this basis since they are not

PLATE I

FIG. 1
Macroscopic photograph of the cut surface of the right and left masseter muscles showing pale focal mottling.

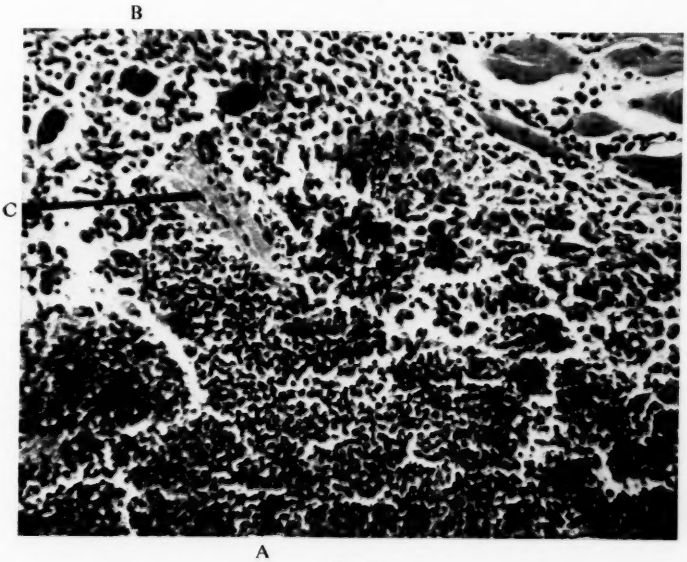


FIG. 2
Photomicrograph of the masseter muscle showing the eosinophil infiltration (A), giant cells (B) and the muscle bundles with sarcolemmal nuclei becoming central (C). Paraffin wax sections, stained H. & E. (X 370).

(Article by Head, Maclennan and Phillips, page 22)

PLATE II

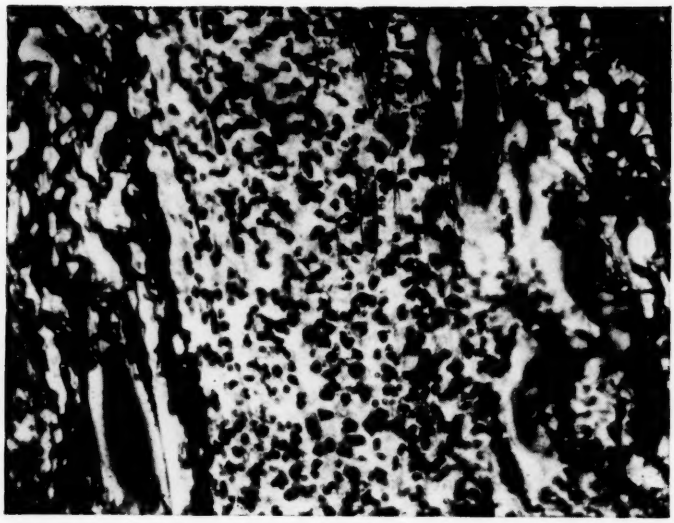


FIG. 3
Photomicrograph of the masseter muscle showing the reticulin network round the muscle bundles but not running into the cellular foci. Paraffin wax sections, Gordon and Sweet's method (X 400).

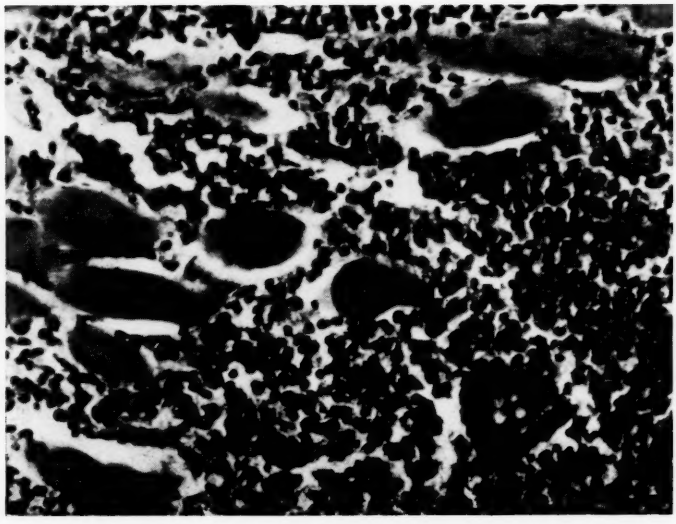
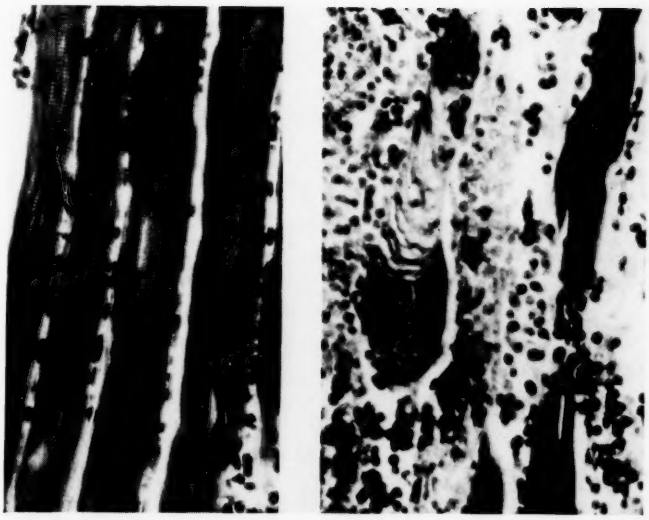


FIG. 4
Photomicrograph of the masseter muscle showing a muscle bundle with central sarcolemmal nuclei and vacuolation of the sarcoplasm; the periphery of the bundle is dark red and condensed compared with the centre of the bundle. Paraffin wax sections, stained H. & E. (X 400)

(*Article by Head, Maclennan and Phillips, page 22*)

PLATE III



A B

FIG. 5

Photomicrographs of the masseter muscle showing:—
(a) Relatively normal muscle bundles with longitudinal and transverse striations.

(b) Abnormal muscle bundles with irregular condensation of the fibres, loss of striation and fragmentation of the sarcoplasmal remnants. Paraffin wax sections, stained Heidenhain's haematoxylin (X 400).

(*Article by Head, Maclennan and Phillips, page 22*)

associated with muscle. The nodules have some similarity histologically to those described by Kunze (1926) under the name of Calcium gout, except that in some of his cases the lesions had a distinct giant cell reaction associated with them and, of course, they occurred in the subcutaneous tissue of the extremities of the limbs.

The hæmosiderin found in the lymph nodes, spleen and liver parallels that described by Harding and Owen (1956) who suggested that this pigmentation was the result of hæmorrhage and muscle breakdown.

Conclusions

The clinical history, blood findings, post-mortem findings and histological examination of a case of Eosinophilic Myositis in a five-year-old female Alsatian are described.

Four cases of atrophy of the head muscles are briefly contrasted with the above. These cases were associated with tuberculosis, distemper chorea, streptococcal arthritis and trauma.

Acknowledgements

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REFERENCES

Clare, M. (1956): *Vet. Rec., 68,* 202-203.
Erichsen, S. (1952): *Nord. vet. Med., 4,* 139-144.
Harding, H. P. and Owen, L. N. (1955): *Vet. Rec., 67,* 460.
Harding, H. P. and Owen, L. N. (1956): *J. comp. Path., 66,* 109-122.
Kunze, A. (1926): *Arch. f. Tierheilk., 54,* 462-478.
Kuscher, A. (1940): *Wien. tierärztl., Mschr., 27,* 177-186.
Moon, C. and Wood, A. G. (1954): *J. Amer. vet. med. Ass., 125,* 312-313.
Robin, V. and Brion, A. (1934): *Rec. Mèd. Vét. 110,* 449-462.
Tamaschke, C. (1955): *Virchows Arch., 327,* 480-489.
Ullrich, K. (1950): *Monatsch. vet. Med. 7 tran. in J. Roy. Army vet. Cps., 25,* 2-3.
Whitney, J. C. (1955): *Vet. Rec., 67,* 1140-1143.
Whitney, J. C. (1957): *Vet. Rec. 69,* 130-131.
Winter, H. and Stephenson, H. C. (1952): *Cornell Vet., 42,* 531-537.
Wirth, D. (1941): *Dtsch. tierärztl. Wschr., 49,* 626-627.

JOHNE'S DISEASE OF CATTLE AND SHEEP IN NEW ZEALAND

Paper presented to the 1957 meeting of the Australian and New Zealand Association for the Advancement of Science.

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JOHNE'S Disease is a chronic enteritis of ruminants caused by *Mycobacterium johnei*. The disease in cattle is worldwide in distribution being reported from the European Continent, the United Kingdom, Sweden, North America, India, Africa Australia and New Zealand. It is prevalent in temperate climates in particular and especially in areas where dairy farming is practised intensely (Doyle, 1956). The discovery of the acid-fast organism in cases of the disease in Europe was made by Johne and Frothingham in 1895. The disease was recognised in the United Kingdom by McFadyean in 1907 who suggested the term Johne's disease. The term " Paratuberculosis " is often used as a synonym. Johne's disease is characterised by gradual emaciation and persistent diarrhœa, a chronic course, and eventual death. Cattle and to a less extent, sheep are affected; goats can be infected experimentally and the disease has been reported in deer.

Stephens and Gill (1937) record that the first proved case of Johne's disease in New Zealand occurred in an imported cow in 1912; in 1928 the occurrence of the disease was confirmed by laboratory examination in a specimen originating in Taranaki; apparently the disease was occurring on several farms but it had not been recognised. Since that time the disease has spread and appreciable losses in dairy cattle have occurred, particularly in the major dairy areas.

Distribution in New Zealand Cattle

The nature of Johne's disease makes studies on distribution and intensity difficult. Farmers often do not report suspected cases, and the methods of diagnosis are not perfect. Many cattle do not die of the disease at the same time; the disease is insidious in nature and losses are spread over a period of time.

In New Zealand Johne's disease is scheduled under a Stock Act and affected cattle must be slaughtered. Compensation is paid. The annual records of condemnations give *some* indication of the distribution and prevalence of the disease. From 1946 condemnations have increased to a marked extent. In 1946 compensation was paid on 94 cattle; the number in 1954 was 513. This is an increase of over 500 per cent in 8 years. This rise may be accounted for by an increase in incidence and by improved diagnostic facilities. This incidence is also indicated in the number of positive ileo-cæcal valve specimens recorded (Chandler, 1957a). The main area affected in New Zealand is Taranaki. The disease is not confined to this area. History suggests that Johne's disease has existed in Taranaki for a considerable period and this site has provided a focus for its spread elsewhere. An appreciable number of cases also occur in the Waikato. Since the war, cases have been reported regularly from North Auckland where there appears to be danger of the disease becoming firmly established. This may be connected with the increase in the

dairy industry in recent years, and new stock may have introduced the disease from the Waikato and from Taranaki. Johne's disease has occurred in such widely distant areas as Hawke's Bay, Wairarapa and Levin and also in the South Island of New Zealand, mainly Southland. These incidents seem to be sporadic and many have occurred in cattle imported from the North Island. In the past ten years, Johne's disease has been confirmed in 38 different counties in New Zealand, including the most important dairying areas of the country. Its distribution may be related to soil and climatic conditions. With its good rainfall, equable temperatures, permanent pastures and intensive dairy farming, New Zealand is particularly favourable towards this infection.

Symptoms and Pathology in Cattle

The symptoms in New Zealand cattle are similar to those elsewhere and consist basically of loss in condition, emaciation and scouring. The onset of symptoms is in many cases gradual, and often the first signs are a change in the colour of the coat and a loss in milk production. The eventual wasting is very marked, especially around the hind quarters; bottle jaw sometimes occurs (Stephens, 1955). In the advanced stages of the disease the beasts become very weak and rarely are capable of reaching an abattoir alive. I have heard no reports of recovery from the disease in New Zealand. Stephens, who has observed the disease in Taranaki for many years, has made the following observations.

" An owner, when wise to the ways of Johne's disease, culls his cows as soon as the slight loss of condition is evident. At present cull cow prices he makes more this way than by waiting for condemnation. Hence, the ones reported are by no means representative of the numbers actually infected in the herd, but are cases that have developed so rapidly that the owner has been caught unawares.

" The commonest age when clinical symptoms are first manifested in Taranaki is between 5 and 7 years. The periods when clinical cases mostly manifest themselves in Taranaki are from June to November (i.e. around the period of calving) with another smaller peak about February . This last is most marked when there has been a fair spell of dry weather. Odd cases break out throughout the year.

Most cases are dead within a year of the onset of scouring. Feed, weather conditions, state of lactation and pregnancy influence the period of survival."

On post-mortem examination the carcase shows emaciation and lesions of the intestine. The latter is thickened, œdematous and sometimes inflamed and corrugated in appearance. As a rule the area of the ileo-cæcal valve is affected, but in many cases the whole of the cæcum and part of the small intestine and colon are involved. Histological examination of the intestine reveals masses of endotheloid cells and, in the majority of cases, numerous Johne's bacilli.

Diagnosis in Cattle

The diagnosis of Johne's disease depends to a great degree on clinical signs. The signs of Johne's disease are simulated in several other conditions and few veterinary surgeons would be content to make a positive diagnosis on clinical signs alone. There is a need for early diagnosis and elimination of the disease in animals incubating the disease but showing no symptoms. These animals may be an important source of infection by acting as " carriers ". Diagnostic methods that

have been used in New Zealand (Chandler, 1956a) comprise fæcal and ileo-cæcal examination, johnin tests and complement-fixation tests.

Smears of fæces, stained by the Ziehl-Neelson method, can be examined for the presence of mycobacteria. Other bacteria can cause confusion and the examiner must have considerable experience before a reliable answer can be given. The methods of examination of the ileo-cæcal and other intestinal specimens are the examination of stained smears, histological examination and culture. The presence of an appreciable number of " acid-fast " bacilli in the smear is one of the most reliable indicators of the existence of the disease.

Opinions are divided regarding the value of johnin tests. In the United Kingdom, various workers such as Dunkin, *et al.* (1941), Minett (1933, 1935), Green (1946), Taylor (1951a) and Hole (1952) are of the opinion that johnin is unsatisfactory, particularly in the later stages of the disease. In the U.S.A., however, some degree of reliance is placed on johnin (Larsen, 1954). In New Zealand, Stephens (1955) believes that johnin is a very useful diagnostic agent, particularly before clinical signs are manifested. It is recognised that there is room for improvement in these present forms of johnin.

Studies on the complement-fixation test in recent years have given great promise. Hole, in the United Kingdom (1952, 1953) has developed a complement-fixation test which, after continuous and large-scale testing, has proved to be one of the best methods of diagnosis available. Investigations in New Zealand (Chandler, 1955) confirm its efficacy and may be summarised as follows. In the clinical stages of the disease the great majority of cases give a positive reaction, while a negative reaction usually eliminated the possibility of the disease. A proportion of the cattle which are not affected with Johne's disease give a positive reaction also. In cattle showing no evidence of the disease, negative reactions were obtained in 70-80 per cent of the cases. There is a fairly high degree of cross-reaction with tuberculosis which limits the use of the test in cattle from herds affected with tuberculosis.

A new form of complement-fixation test has been designed and developed in New Zealand (Chandler, 1956b). This micro-test offers advantages over the Hole test in easier and faster technique and smaller quantities of reagents. The test is carried out using a perspex tray instead of tubes and racks; the test proper takes 1¾ hours compared with the 3½ hours of the Hole test. This micro-test would appear to be particularly suitable for use at regional centres. Studies at Wallaceville have indicated that the micro complement-fixation test is very comparable with the Hole test and may eliminate some of the " false-positive " results. Under the circumstances of the investigations, the micro-test was 84 per cent efficient in confirming Johne's disease in suspected clinical cases and 77 per cent efficient in identifying those free from the disease, even when these suspects could not be guaranteed free of tuberculosis.

Under New Zealand conditions, a veterinary surgeon who suspects the disease on clinical grounds, submits a fæces sample and a serum sample for examination. A positive fæces result indicates the presence of the disease, but a negative result is of little value. A positive complement-fixation test indicates the presence of the disease, while a negative test, in the majority of cases, excludes it. A johnin

test could be made but its result should be considered only in conjunction with the other evidence. It is after consideration of clinical signs, farm history, differential diagnosis, fæcal examination, complement-fixation test, and possibly johnin test, that a veterinary surgeon makes his diagnosis. The diagnosis of pre-clinical cases presents more difficulty. The evidence suggests that it would not be advisable to condemn any animal merely on the basis of a positive complement-fixation test or positive johnin test.

Treatment of Johne's Disease in Cattle

No effective treatment of Johne's disease is known. Various workers (Larsen, *et al.* (1950); Deans Rankin (1953); Williams Smith (1954)) have investigated the use of antibiotics *in vitro* and *in vivo*. Although certain antibiotics had action against *M.johnei in vitro*, none was found effective against the clinical form of the disease. Palliative treatment with intestinal astringents have been undertaken in New Zealand by Stephens (1955). It did not retard the clinical progress of the disease.

Control of the Diseases in Cattle

The methods of control available for Johne's disease in cattle include diagnosis and destruction of affected beasts, fencing, elimination of stagnant water, separation of calf from dam, movement control and vaccination. A considerable measure of control, in regard to general hygiene and nutrition, is in the farmers' own hands (Matthews, 1947). Vaccination offers a promising method of control in New Zealand.

A vaccine for the prevention of Johne's disease in cattle was evolved by Vallée and Rinjard in France in 1926. This vaccine consisted of a live unattenuated bovine strain of *M.johnei*, suspended in liquid paraffin to which powdered pumice was added. Inoculation resulted in the formation of a fibro-caseous nodule in which the organism persisted.

The vaccine has been used extensively in France but owing to the nature of the disease, and the war, analysis of the efficacy of the vaccine had to be made on a broad rather than a detailed basis. By 1941 more than 250,000 cattle had been vaccinated (Vallée and Rinjard, 1941); the vaccine appeared " safe and generally efficacious ".

Experiments with the vaccine in the United Kingdom were made by Doyle (1945), who demonstrated that it is innocuous, and suggested a trial of the vaccine in United Kingdom herds. In 1946 a field trial was initiated in tuberculosis-free herds suffering from Johne's disease. Controls within the herds were not available but adult cattle in the herds at the commencement of the experiment were not inoculated and the results were judged on subsequent herd history. The available results (1954) of the trial indicate that the average annual incidence is reduced by use of the vaccine.

A trial of the vaccine in New Zealand was instituted in 1948 by Buddle (1956) in co-operation with district veterinary surgeons. The vaccine was prepared in the first instance at Wallaceville but over the past few years the Weybridge vaccine has been used. 3-5 herds were selected in the Taranaki district and 3 in the Waikato district which had suffered heavily from Johne's disease. All the calves born on these properties in this and subsequent years were vaccinated during the

first month of life and not revaccinated subsequently. Results were judged on the subsequent history of the infected herds. Unvaccinated adult cattle continued to die of Johne's disease on the chosen properties, indicating that the vaccinated cattle were exposed to a heavy challenge. The nodules produced at the site of vaccination persisted for many years. One owner withdrew from the experiment completely because of these reactions. Arrangements were made to examine the ileo-cæcal valve of every animal in the vaccinated herds which died or was slaughtered subsequently. None of the vaccinated animals have to date showed clinical signs of Johne's disease. One ileo-cæcal valve from a heifer, culled in good condition two years after vaccination, showed that infection was present. No claims have been made that the vaccine prevents multiplication of *M.johnei* in cattle completely but the accumulated results indicate that it prevents the *clinical* manifestation of the disease in a great proportion of the cases. Stephens (1955) reports that a further indication of the efficacy of vaccination was shown by the fact that the number of calves reared in some of the vaccinated herds had to be reduced substantially, as the herds began to suffer from a plethora of replacements.

Allergic Tests on Cattle Vaccinated Against Johne's Disease

Vaccination against Johne's disease has the disadvantage that vaccinated cattle may react to the tuberculin test; vaccination may interfere with tuberculin eradication.

Early French work by Vallée and Rinjard (1926) had indicated that vaccination resulted in no mammalian tuberculin reaction, but Doyle (1945) in the United Kingdom, used improved tuberculins (intradermal neck test) and showed that a majority of vaccinated calves gave positive reactions 1-2 months after vaccination. Buddle (1953) in New Zealand has obtained strong positive mammalian tuberculin reactions in experimental calves 3 months after vaccination. Stephens (1955) observed vaccinated New Zealand cattle and reported that cattle one year after vaccination, gave strong johnin reactions, slightly less avian tuberculin reactions and moderate tuberculin reactions. He believed that any confusion regarding the positive mammalian reaction could be solved by examination for the vaccination nodule and by conducting a comparative test. Doyle (1953) has shown that goats vaccinated against Johne's disease and *infected with tuberculosis* may give higher avian tuberculin than mammalian tuberculin reactions. A comparative test might fail therefore to diagnose tuberculosis in vaccinated cattle.

It is hoped to extend the use of the vaccine in New Zealand but the possible interference with any future scheme for the eradication of tuberculosis has prevented this up to the present time. Investigations were designed to evaluate the reactions produced in vaccinated cattle by the intradermal neck test, the caudal fold test and a thermal test (Chandler, *et al.*, 1957). The intradermal neck test, using avian and mammalian tuberculins, together with johnin, was chosen so as to elaborate on Stephens' observations. The caudal fold test was studied since this is the test in general field use in New Zealand. The thermal test used was the "short thermal tuberculin test" described by Gregory (1949); this test has been used on a fairly extensive scale in Australia.

The results with the intradermal neck tests confirmed the opinion formed in

other countries that the high proportion of positive mammalian tuberculin reactions would exclude the neck test for tuberculosis in a vaccinated herd. Similarly, the results with the caudal fold test indicate that this tuberculin test would not be suitable for the eradication of tuberculosis in vaccinated herds. This is of particular importance in New Zealand, where the caudal fold test is used commonly.

The studies with the short thermal test show that in vaccinated herds where tuberculosis is uncommon, the thermal test produces a far lower proportion of positive reactions than any of the other tests. The positive reactions given by the thermal test may have been due, at least in part, to tuberculosis in these vaccinated cattle. In vaccinated herds where tuberculosis was prevalent, the short thermal test produced an appreciable number of positive reactions but it is likely that tuberculosis itself was responsible for the reaction in many cases. Studies on thermal response in young cattle indicated that vaccination induces some sensitisation to subcutaneous tuberculin; but the circumstances of the studies do not enable one to conclude that *adult* cattle would give positive results to the standard short thermal tuberculin test owing to the effect of the vaccine alone. It is evident that the short thermal test is the only one of those studied which is worthy of further investigation in regard to its use in vaccinated herds.

Johne's Disease of Sheep in New Zealand

Johne's disease of sheep in New Zealand was reported by Williamson and Salisbury (1952), who confirmed its presence on a sheep property in the South Canterbury district of South Island. Since that date the disease has been diagnosed by Armstrong (1956), and confirmed at Wallaceville, in 13 further flocks in South Canterbury and 2 in the adjacent area of Ashburton. Armstrong states that there is no evidence that the disease has appeared suddenly and has spread to all the properties in the last few years. The properties concerned have probably been infected for several years but the mortality has on most farms has been so low as to excite little interest. The diagnosis in 1952 stimulated interest in the disease and as a result the disease was recognised on the several properties. It is not unlikely that the disease exists elsewhere in New Zealand but has not so far been diagnosed. The incidence of the disease in affected flocks is usually low but has been as high as 5 per cent of breeding ewes on one property.

Diagnosis of the Disease in Sheep

The history associated with the disease of sheep in New Zealand is usually one of progressive emaciation and death in breeding ewes. Most confirmed cases have occurred in 4-year-old ewes. The symptoms are predominantly those of gross emaciation; scouring does not occur in all cases.

The post-mortem appearances are those of emaciation, together with intestinal lesions. The latter are seen as thickenings of the distal portion of the ileum, the cæcum, and the proximal portions of the colon. The lymph vessels on the intestinal surface are, in many cases, very prominent. Smears of the affected gut, stained appropriately, show masses of bacilli; histological specimens show massive invasion of the mucous membrane and submucosa with the bacilli.

Taylor (1951b) has shown that in the northern hemisphere three varieties of *M.johnei* can cause the disease in sheep; the classical bovine variety, a pigmented

variety and an Icelandic type which is very difficult to culture. No pigmented lesions have been seen in affected New Zealand sheep and investigations are in progress to decide which of the other two varieties is the causal organism.

In sheep, the laboratory diagnostic methods used are very similar to those in cattle. Examination of the fæces appears to be more reliable in sheep, possibly because there is less scouring in affected sheep than in affected cattle. The impression is gained also that the number of organisms present in sheep infections is relatively higher than in cattle. Fæces examination nevertheless detected only 55 per cent. of positive cases in these studies, and a more efficient diagnostic method is required. Ileo-cæcal specimens from sheep are satisfactory but adequate samples of affected ileum should be examined, since it is noticed that the disease is often most marked in this portion of the bowel. Johnin is seldom used in sheep. Sigurdsson, *et al.* (1945) have described a complement-fixation test for Johne's disease of sheep in Iceland. It was a very useful diagnostic aid. Studies (Chandler, (1956b) have been carried out in New Zealand to ascertain the use of the micro complement-fixation test in affected flocks. With this test ovine sera can be tested at the same time as bovine sera. The micro-test appeared very promising for use in sheep; in clinically suspected sheep the test is *at least* 80 per cent effective in detecting positive cases and *at least* 83 per cent effective in proving that suspects are free of the disease.

Control of the Disease in Sheep

The methods of control available for Johne's disease in sheep are similar to those used in cattle. Vaccination studies have been instituted to make a vaccine available for highly affected properties and as a safeguard should the disease position become worse.

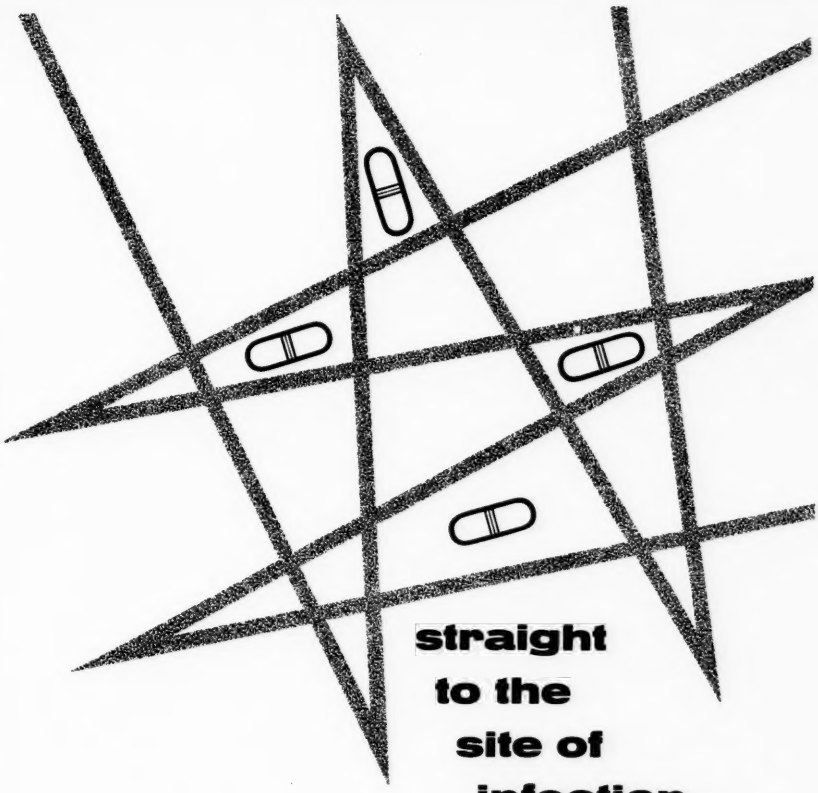
Sigurdsson, *et al.* (1949, 1950, 1952) have used with success a heat-killed bovine strain of *M.johnei* against the disease of sheep in Iceland. Studies on a similar vaccine, and on one modified by grinding the bacilli, have begun in New Zealand (Chandler, 1957b). Laboratory studies have shown that vaccinated sheep are in no way indisposed by the local nodule produced, and that complement-fixing antibodies are produced together with a high level of sensitivity to johnin. A field trial with the vaccines has begun on an affected property in South Canterbury and results of this are awaited.

In conclusion it may be said that the importance of Johne's disease, in both cattle and sheep in New Zealand, is recognised. The disease causes appreciable losses to basic livestock production, and constitutes a hazard to successful export marketing of the country's stock. Research on the problem has been shown to yield useful information and results; the disease will not be vanquished without great efforts from all concerned.

Summary

Johne's disease of both cattle and sheep exists in New Zealand. The disease in cattle is considered of major importance; the disease in sheep has so far resulted in few losses but it constitutes a potential menace to the sheep industry.

The diseases in cattle was in all probability imported into Taranaki in infected but apparently healthy cattle. The disease has spread and appreciable losses in



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dairy cattle have since occurred, particularly in Taranaki and Waikato, the major dairying areas.

Diagnosis is based on symptoms, history and a complement-fixation test. Confirmatory diagnosis is made by post-mortem examination. Johnin has been used as a diagnostic agent but opinions differ as to its value. The use of the Hole complement-fixation test has been investigated. The results confirm the value of the test and show it to be one of the best available for Johne's disease in its clinical stages. A micro complement-fixation test has been developed which offers advantages over the Hole test in ease of technique and economy of technical staff. Results obtained with this micro test compare very well with those of the Hole test and it has been adopted as the routine test in New Zealand. Control of the disease has been the subject of a field vaccine trial over a period of years. Vaccination has resulted in elimination of clinical cases of the disease on the farms concerned, and appears to be the best method of control available. Extension of the use of the vaccine is desirable but its effect on any subsequent tuberculosis eradication scheme must be considered, since vaccinated cattle react to the tuberculin test.

The disease occurs in sheep on properties contiguous with the Canterbury Plains. The first case was diagnosed in 1952 and since that date the disease has been confirmed on 15 properties. There is no evidence that the disease has appeared suddenly and spread to all these properties in the last few years. Mortality is low, the disease occurring mainly in breeding ewes. The symptom of diarrhœa is not commonly seen, but emaciation is marked and the disease is fatal. Diagnosis in life is based on history, symptoms, examination of the fæces and the micro complement-fixation test. Vaccines are being studied and a trial begun on an affected property.

Johne's disease of cattle and sheep in New Zealand is of importance, not only to the basic livestock production of the country, but also to the export market for this country's stock.

REFERENCES

Armstrong, M. C. (1956): *N.Z. vet. J., 4,* 56.
Buddle, M. B. (1953): Private Communication.
Buddle, M. B. (1956): Paper read to Annl. Conf. of N.Z. vet. Assoc., 1956.
Chandler, R. L. (1955): *N.Z. vet. J., 3,* 145.
Chandler, R. L. (1956a): *Ibid., 4,* 121.
Chandler, R. L. (1956b): *Vet. Rec., 68,* 819.
Chandler, R. L. (1957a): *N.Z. vet. J., 5,* 1, 20.
Chandler, R. L. (1957b): *Ibid., 5,* 2, 39.
Chandler, R. L., Macdonald, J. H., Caldwell, D. W., Griffiths, C. V. W. and Isaacs, C. E. (1957): *N.Z. vet. J., 5,* 1, 24.
Deans Rankin, J. (1953): *Vet. Rec., 65,* 649.
Doyle, T. M. (1945): *Ibid., 57,* 385.
Doyle, T. M. (1953): *Brit. vet. J., 109,* 275.
Doyle, T. M. (1956): Paper presented to 74th Annl. Conf. Brit. vet. Assoc.
Dunkin, G. W., Glover, R. E. and Minett, F. C. (1941): *J. Hyg., 41,* 297.
Green, H. H. (1946): *Brit. vet. J., 102,* 267.
Gregory, T. S. (1949): *Austral. vet. J.,* 25, 138.
Hole, N. H. (1952): *Vet. Rec., 64,* 601.
Hole, N. H. (1953): Rep. No. 287 from XXI Session, Int. Bureau Epizoot. Dis.
Johne, H. A. and Frothingham, L. (1895): *Dtsch. Z. Tiermed., 21,* 438.
Larsen, A. B., Vardaman, T. H. and Groth, A. H. (1950): *Amer. J. vet. Res.,* II, 374.
Larsen, A. B. (1954): *Proc. Amer. vet. Med Assoc.* Annl. Conf.
Matthews, H. T. (1947): *Vet. Rec., 59,* 397.
Minett, F. C. (1933): *Vet. Rec., 13,* 1074.
Minett, F. C. (1935): *J. comp. Path., 48,* 125.

Ministry of Agriculture (1954): "Report on Animal Health Services in Great Britain." London, H.M.S.O.
McFadyean J. (1907): *J. comp. Path., 20,* 48.
Sigurdsson, B., Vigfussion, H. and Theodore, S. (1945): *J. comp. Path., 55,* 268.
Sigurdsson, B. and Trygvadottir, G. (1949): *J. Bact., 58,* 271.
Sigurdsson, B. and Trygvadottir, G. (1950): *Ibid., 59,* 541.
Sigurdsson, B. (1952): *J. Immunol., 68,* 559.
Stephens, E. H. (1955): Private communication.
Stephens, E. H. and Gill, D. A. (1937): *N.Z. J. Agric., 54,* 1.
Taylor, A. W. (1951a): *Vet. Rec., 63,* 776.
Taylor, A. W. (1951b): *J. Path. Bact., 63,* 333.
Vallee, H. and Rinjard, P. (1926): *Rev. Gen. Med. vet., 35,* 1.
Vallee, H., Rinjard, P. and Vallee, M. (1941): *Bull Acad. Med., 125,* 195.
Williams Smith, H. (1954): *J. comp. Path., 64,* 15.
Williamson, G. J. and Salisbury, R. M. (1952): *N.Z. vet. J., 1,* 15.

FARM LIVESTOCK RESEARCH STATION

Stock, Near Chelmsford, Essex

History

THE Animal Health Trust was founded in 1942 and research work on animal diseases was initiated almost immediately. For some time this work was carried on at various universities by the award of fellowships and at Houghton in Huntingdonshire, where laboratory and other facilities were provided along side the Poultry Research Station.

As the activities of the Trust expanded, more accommodation became necessary. In 1955, the Poultry Station was reorganised as a grant-aided establishment in association with the Agricultural Research Council, and more commodious premises had to be found for the Livestock Section.

At this critical moment the Trustees of the Boreham Institute (then under the chairmanship of the late Lord Perry) made the generous offer which was to lead to the transfer of the A.H.T. Farm Livestock Research Station to Stock in Essex.

Under the terms of this offer the Boreham Institute undertook:—

(a) to erect at their own expense, on land surrounding Lilystone Hall the necessary laboratories and other scientific buildings required by a modern research station and

(b) to lease the whole property to the Animal Health Trust on a long-term basis and at a "peppercorn" rental.

The offer was accepted with gratitude by the Trust and the foundation stone of the new station was laid by His Grace the Duke of Norfolk (President of the Trust) on March 1st, 1956. Erection of the buildings was completed, and the staff entered into occupation in the summer of 1957, research work having been carried on meanwhile in temporary accommodation in Lilystone Hall.

The new station was opened by H.R.H. Prince Philip on Friday, December 6th, 1957.

The total cost of the new station, including scientific equipment, is approximately £100,000. Part of the new equipment has been placed at the Trust's disposal by the Milk Marketing Board. The A.H.T. and the M.M.B. are co-operating on certain research projects.

The total area of the property is about 60 acres. This includes some 40 acres of pasture and park land and about 10 acres of ornamental gardens surrounding Lilystone Hall. The latter are an outstanding feature of the estate and will continue

to be maintained by the Boreham Institute without cost to the Trust. The Hall itself is available to the Trust for offices, conference or lecture room and flats for some of the scientific workers.

The laboratories and other research buildings are situated on an island site of about 16 acres enclosed by a ring fence and surrounded by hard roads on all sides. Space is available to allow for future expansion and, in designing the present buildings, the architects have made provision for additional accommodation to be erected as the need arises. As it stands now the station comprises:—

Main Building.—This consists of a central block containing on two floors, the administrative offices, boiler house, media and preparation rooms, photographic darkroom, stores, working and general laboratories. The adjoining research wing, also of two floors, consists of ten laboratories plus smaller rooms, those on the lower floor being devoted to bacteriological and pathological research and those above to biochemistry and nutritional work.

Large-animal House.—The accommodation consists of seven loose-boxes, preparation room and small food store. There is also a covered concrete exercising yard. This building follows the pattern used by many research centres including the A.R.C. Station at Compton, and the boxes are suitable for any class of farm stock.

Houses for Smaller Stock.—These twin buildings—as yet incomplete—are intended for housing small experimental animals such as guinea pigs, rats, mice and rabbits, and for small farm animals such as pigs, lambs and calves. The buildings are double-walled with insulated roofs and there are points for electric heating. Each has a preparation and washing room and small food store.

The Piggery.—A wooden structure presented to the Trust while the station was at Houghton was transferred to Stock where it has been re-erected on a concrete deck. This will be used for short-term nutritional-cum-bacteriological experiments.

Post-Mortem Room.—Contains a post-mortem area and benches on which material can be examined. There is also a " butcher's type " refrigerator for storing specimens and a travelling hoist capable of handling all types of stock.

Incinerator.—Animal material, bedding, etc. which must be disposed of by burning can be dealt with in a modern coke-fired and electric forced-draught incinerator.

Sewage Plant.—All effluent from the animal houses and laboratories is passed through a special sewage plant installed on the site.

Personnel

In its present stage of development, the station has recruited only a comparatively small staff, 24 in all—scientific, administrative, technical and clerical. The scientific personnel consists of:—

K. C. Sellers, B.Sc., Ph.D., M.R.C.V.S., D.V.S.M., (Director).
H. Williams Smith, D.Sc., B.Sc., Ph.D., F.R.C.V.S.Dip.Bact.(London).
A. L. Ogden, B.Litt., M.R.C.V.S., D.V.S.M.
G. C. Ashton, B.Sc.
G. Skulski, M.R.C.V.S.
W. E. Crabb, A.I.M.L.T.

As the station progresses, so the staff and accommodation will be increased to

enable research problems to be tackled from the pathological, bacteriological, virological and parasitological angles, along with biochemical and nutritional studies. Diagnostic and clinical services will be developed and additional facilities provided for field surveys into disease on farms and *ad hoc* problems.

General Research Policy

The research programme has been planned to meet the urgent needs of the livestock industry. Field surveys are being carried out to discover which of the many diseases and conditions cause the greatest economic loss to the farmer, thus neither time, money nor materials are wasted on problems of little immediate practical importance. Attention is focused on the diseases of young and growing stock, and on preventing the reproductive wastage which causes so much financial loss among all classes of animals.

Current Investigations

In cattle, useful work has already been done on Johne's disease. The station is engaged also in an exhaustive survey into the incidence and causative factors of bovine infertility. This is carried out in collaboration with the Milk Marketing Board. Arrangements have been made to study a random sample of some 200 breeding herds in East Anglia, thus providing a cross-section of some three to four thousand head of cattle for investigation. All departments of the station are co-operating in this task.

Factors involved in scour conditions in calves and young pigs are being investigated in the field. Already a technique has been devised whereby, from an examination of dung samples, an assessment can be made, within a period of eight hours, of the best treatment for affected animals. These investigations reveal that the bacteria involved in the scouring condition may develop a resistance to antibiotics. It is too early yet to decide on the significance of this finding, and work on the subject is continuing.

In the biochemical department, the main emphasis of present work is on an investigation into the part played by iodine and vitamin A in the growth and disease of pigs. At the same time a full-scale study is being made on the blood serum of animals, more especially cattle, in order to explore the possibility of a link between blood types and desirable economic characteristics such as milk quality. With the full co-operation of the Milk Marketing Board, an extensive range of blood samples from herds included in the Board's progeny-testing schemes are being tested. In addition samples have been obtained from bulls standing at M.M.B. and private insemination centres. These examinations indicate that cattle can be classified into five clearly defined groups according to their blood serum protein patterns. Results already obtained in this investigation have given a very satisfactory picture of the distribution of types in the different breeds in this country and strongly suggest that accumulated data will lead to improved genetical knowledge as well as to better control of disease.

Sheep are receiving their share of attention at the station, and emphasis is placed on the problems of losses among breeding ewes and young lambs. A small flock of Kerry Hill (Wales) breeding sheep has been installed at Stock for these and other experimental purposes. Last season an " on-the-spot " survey of breeding losses in East Yorkshire was carried out by a member of the Trust's scientific staff tem-

porarily stationed in that area. The data obtained (already fully reported in the agricultural and scientific Press) will be used as the basis for further studies of the subject at the station during the forthcoming winter and spring.

As for pigs, the Trust is maintaining close collaboration with the National Pig Breeders' Association in order to ascertain the most profitable way in which the station can be used to help this important industry. Close liaison has been established with several large breeding farms in the neighbourhood to enable an objective survey to be made of practical problems. The pressing need for more research into pig diseases, is appreciated, and it has been decided to set up a special experimental unit at the station so that investigation of pig problems in the field can be correlated with controlled scientific observations in the laboratory.

In addition to its fundamental research work, the station provides clinical and diagnostic services for the farmer. Cases and specimens are dealt with through the farmer's veterinary surgeon.

The Trust has the responsibility for finding the money to pay for staffing and day-to-day running costs. Its entire income depends upon voluntary contributions. The work that can be undertaken at the Livestock Station depends in great measure upon adequate financial support. It is estimated that for the next year or two the annual cost will be about £40,000 a year. As the station develops and its activities expand this annual budget will increase to about £100,000 per year. This figure is large but it is infinitesimal compared with the annual turnover of the industry which the station exists to serve.

There is a choice of several easy ways in which the farmer can make his contributions—either by becoming a regular subscriber by means of the usual banker's order or by joining, according to his particular branch of husbandry, one or other of the " pay as you earn " contributory schemes run on behalf of the Trust by the Milk Marketing Board, the Fatstock Marketing Corporation or certain Livestock Auctioneers. Under these schemes arrangements are simply and conveniently made for certain specified deductions to be made regularly at stated intervals from moneys due to the farmer from the sale of his products. This method is finding increasing popularity since farmers are thereby relieved of all further trouble in the matter, the marketing organisation concerned shouldering all the clerical responsibility for passing the money thus collected over to the Trust.

All contributions made rank as legitimate business expenses from the Income Tax point of view.

REVIEWS

VETERINARY PATHOLOGY, by H. A. Smith and T. C. Jones. 1957. London: Henry Kimpton. Pp. 959. 661 illustrations on 263 figures and a colour plate. £6 10s. net.

THIS book cannot fail to take its place as a classic textbook on its subject. The senior author is Head of the Department of Veterinary Pathology, School of Veterinary Medicine, A. & M. College of Texas; the junior author had the invaluable experience of being Chief of the Veterinary Pathology Section of the Armed Forces Institute of Pathology in Washington. The book deals broadly with the ætiology, the pathogenesis and the macroscopic and histological lesions

of diseased conditions, chiefly, although not exclusively, those of domestic animals. General systematic and special pathology is therefore covered although it is impossible, even in the scope of some 900 pages, to give a complete survey of veterinary pathology. The authors have contrived to present in an attractive fashion a lot of detailed information about most of the important disease conditions of domestic animals; they have not tried to deal fully with all avian pathological conditions. Apart from its utilitarian value, veterinary pathology is presented as an absorbingly interesting subject and this interest is communicated to the reader. The style is pleasingly didactic and encouragingly authoritative.

The first part of the book deals in some 200 pages with general pathological conditions, such as degenerations, infiltrations and inflammation; here and elsewhere the authors pay due attention to relevant human conditions and their treatment of what can sometimes seem a dull part of the subject is refreshingly lucid and "veterinary" in its outlook. In the chapter on neoplasia, the authors have constructed a most useful table, drawn from selected published surveys and from cases at the Texas school and the Registry of Pathology, showing the location and types of 7,052 tumours of domestic mammals—5,315 in dogs, 385 in horses, 954 in cattle, 121 in sheep, 137 in pigs and 140 in cats.

Next, in about 400 pages, an account is given of the pathology of diseased conditions due to viruses, rickettsiæ, bacteria, fungi, spirochætes, protozoa, helminths, arthropods, poisons and metabolic disorders. The conditions dealt with are not confined to those occurring in the U.S.A., although the authors seem to be more fully at home with these "native" conditions. Of particular interest are the diagrams of the distribution of lesions in neurotropic virus diseases, and the account of equine viral arteritis, while workers in this country will appreciate the full account of coccidiodomycosis, haplomycosis, cryptococcosis, histoplasmosis, toxoplasmosis and eperythrozoonosis, and of the pathological effects of ionizing radiations. The chapter on disease due to poisons is a very useful one: the authors have grouped the various poisons according to some outstanding lesion which would offer a clue to the diagnosis. There is much information here, as elsewhere in this book, that may be new to workers outside America.

The last 300 pages of the book deal with systematic pathology; this is the part of the book which could most usefully be expanded in the next edition, but even so it gives an exhaustive survey of the more important disease processes affecting the different organ systems of the domestic animals.

The book is illustrated profusely. There are a large number of photomicrographs. Most of the pictures are good, some are excellent—Fig. A, p. 171, is the poorest. The illustrations alone would commend the book to the student as an aid to diagnosis in laboratory and post mortem room. A further good feature is the provision of a list of selected references (totalling over 1,900) at the end of each chapter. There is a full index amounting to 30 pages.

In a work of this kind there are bound to be some things with which the reviewer would disagree. Thus, the account of bone diseases is not entirely satisfactory from the point of view of classification of the osteodystrophies. Necrobiosis is not a synonym of necrosis (p. 16). The authors appear to confuse cholesteatomas with cholesterinic granulomas (pp. 209, 897). The word "dopa" was apparently not coined from the "key letters" of dihydroxyphenylalanine, but from the Continental equivalent dioxyphenylalanine (p. 60). Catarrhal inflammation and mucous

inflammation are not synonymous (p. 131). *Cl.septique* is not the cause of enterotoxæmia in sheep (p. 100). Canine mixed mammary tumours are not confined to females (p. 197). In the reviewer's experience, squamous cell carcinoma affects the tongue of the cat with some frequency, but practically never that of the dog (p. 741), and similarly the cat's œsophagus is not an uncommon site of carcinoma formation (p. 744). It is a little strong to say that pancreatic disease is extremely rare in domestic animals (p. 776), bearing in mind its relative frequency in the dog. It is doubtful if the concept of " alterative inflammation " (p. 133) is didactically advantageous.

A few misprints have been noted, including: Black's (disease) (pp. 15, 932); Celsius (p. 118); Hodgkins' (disease) (pp. 138, 941); (T.) crassicolis (p. 215); Oestris (ovis) (p. 250); pruritis, Trabberkrankheit (p. 259); Cole (in dealing with L.C.L. bodies) (p. 318); diptheria (p. 376); viens (p. 482); hypoglycemia (in milk fever) (p. 623); eptopic (p. 836); diskeratosis (p. 945); metastatis (p. 946). The work of Salmon and Smith on hog cholera is wrongly dated 1855 (p. 292).

The book can be recommended with confidence to veterinary students and field and laboratory workers. It should also be of interest to medical pathologists, and to those working with laboratory animals such as monkeys. Its price seems high but, even allowing for the adverse dollar exchange, it is very good value indeed.

SHEEP HUSBANDRY AND DISEASES, by Allan Fraser, D.Sc., M.D., and John D. Stamp, D.Sc., M.R.C.V.S., F.R.S.E. 1957. London: Crosby Lockwood & Son, Ltd. Pages xii and 444. 93 illustrations. Price 35s.

THIS textbook, now in its third revised edition and with a new section, can be recommended to all those interested in sheep, whether they be stockmen, veterinary surgeons or students. The new addition on sheep diseases enlarges the book considerably on past editions, and it is good to note that two well-known authorities have combined together to emphasize the relationship between husbandry and disease. The first seven chapters deal comprehensively with all aspects of sheep breeding, the production of mutton, wool and milk, the whole picture of nutrition and finally general sheep husbandry. The last five chapters concern diseases of sheep. The facts are presented in a simple and lucid manner and yet comprehensive enough to avoid criticism.

The book is illustrated very well, although more photographs in the section on diseases would be appreciated by most readers.

PHARMACEUTICAL RESEARCH IN I.C.I., 1936-1957. Imperial Chemical Industries. Limited. Pp. 88.

IMPERIAL CHEMICAL INDUSTRIES have published this book to commemorate the opening of their new research laboratories. The first part of this handsomely produced volume summarises the history of pharmaceutical research by Imperial Chemical Industries. In the third decade of the present century a small beginning was made as an off-shoot of the Dyestuff Group. The medicinal chemical section really developed from 1936 onwards. Their contribution to the war effort of this country was enormous, particularly in the development and supply of the antimalarial products and penicillin. The veterinary side of I.C.I. activities began about 1942 when it was realised that no distinction could be drawn between medical and veterinary objectives.

The second part of the book describes the new laboratories at Alderley Park

in Cheshire, and is illustrated profusely with photographs and plans. The laboratories, which comprise an animal breeding house, experimental animal houses, biological and chemical laboratories, are most up to date and very carefully designed.

Finally an outline is given of the organisation of the Research Department. The Chemical Group has sections dealing with antiparasitics, organic diseases, general chemistry, biochemistry, formulation, antibiotics and analysis. The Biological Group has sections dealing with virology, bacteriology, pathology, pharmacology, physiology, parasites and statistics. There is some deliberate overlapping between these sections.

Amongst those drugs developed by I.C.I. are:

"Kemithal"—Thialbaritone B.P.C.
"Sulphamezathine"—Sulphadimidine B.P.
"Avlosulfon" "Udolac"—Dapsone B.P.C.
"Anavenol"—Betanapthoxyethanol B.Vet. C.
"Paludrin"—Proguanil B.P.
"Antrycide"—Quinapyramine B.Vet.C.
"Mysoline"—Primidone B.P.
"Hibitane"—Chlorhexidine.
"Fluothane"—Halothane.

Reading this book one realises how much veterinary surgeons owe to the initiative and foresight of the large industrial organisations.

NOTICE

AGRICULTURAL RESEARCH COUNCIL

Testing of Veterinary Medicines

THERE has been a recurrent suggestion that in view of the many substances which are now being marketed for the prevention, cure and control of animal diseases, there is need of some scheme whereby the value of particular substances may be tested under field conditions by an impartial body. As the result of discussions with representatives of the British Veterinary Association and of the Association of British Pharmaceutical Industry, the Agricultural Research Council have now initiated a scheme by which tests can be carried out under the authority of a Veterinary Therapeutic Testing Board appointed for the purpose by the Council.

It is intended that tests shall normally be made on the initiative either of a manufacturer of the substance concerned or of an organisation representing the veterinary profession, and that they shall be carried out by practising veterinary surgeons, who will be invited to co-operate, in accordance with a plan prepared by a group representing those interested in the test.

The Board, of which the Chairman is Mr. M. Noble, includes members appointed by the Council after consultation with the Royal College of Veterinary Surgeons, the British Veterinary Association, the Pharmaceutical Society, the Association of British Pharmaceutical Industry and the Medical Research Council.

Copies of the scheme and of a leaflet stating the general conditions governing tests can be obtained from the Secretary, Veterinary Therapeutic Testing Board, Agricultural Research Council, Cunard Building, 15 Regent Street, London, S.W.1, to whom any communications should be addressed.

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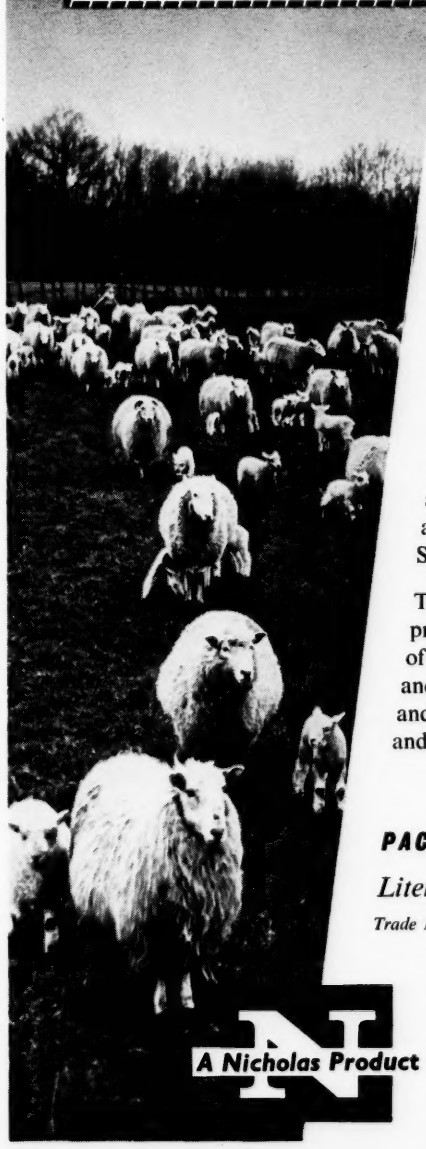

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The Senate will shortly proceed to an appointment to the CHAIR OF VETERINARY MEDICINE or the CHAIR OF VETERINARY SURGERY. The present occupant of the Chair of Veterinary Science, Professor R. M. C. Gunn, will retire in 1958 and the Chair under this title will not be filled. Until such time as it is found possible to fill both the Chair of Veterinary Medicine and the Chair of Veterinary Surgery, the person appointed to either Chair will be expected to exercise supervision over the other field.

Salary will be at the rate of £A3,000 per annum, plus cost of living adjustment (£70 men, £53 women). The current scale of salaries is under revision. There is a normal retiring provision under the Professorial Superannuation Scheme and in addition a pension of not less than £A400 per annum upon retirement after the age of 60 years. At present the amount is £A700 per annum.

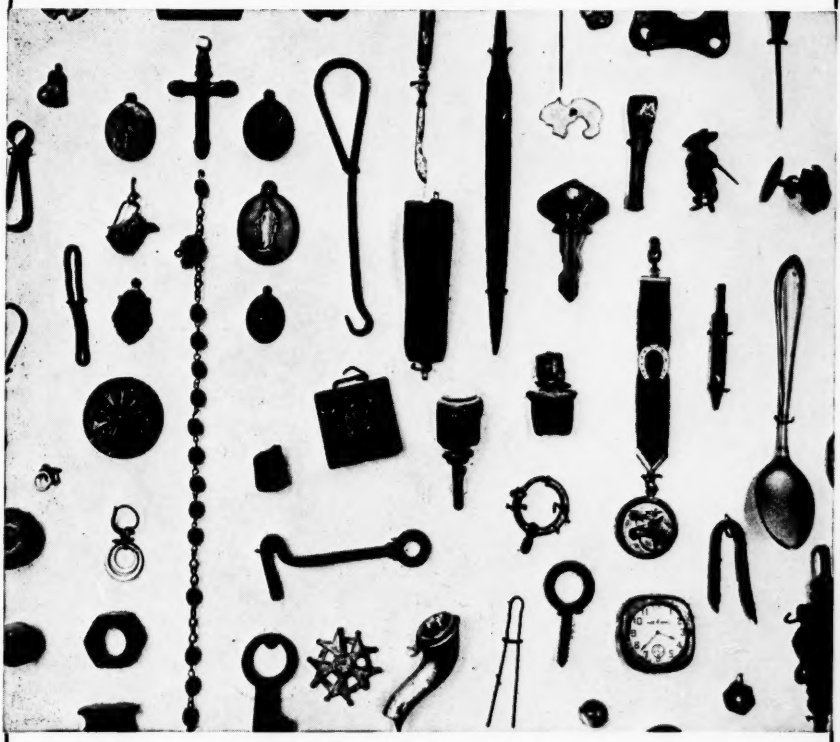
Under the Staff Members' Housing Scheme, in cases approved by the University and its Bankers, married men may be assisted by loans to purchase a house.

The Senate reserves the right to fill the Chair by invitation.

A statement of conditions of appointment and information for candidates may be obtained on application to the Secretary, Association of Universities of the British Commonwealth, 36 Gordon Square, London, W.C.1.

Applications close, in Australia and London on **28th February, 1958.**

THE THINGS A COW WILL EAT



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Notices

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The British Veterinary Journal is intended for the publication of papers on original work and reviews on all aspects of veterinary science and cognate subjects.

Manuscripts should be sent to the Editor, *The British Veterinary Journal*, 7 and 8 Henrietta Street, London, W.C.2, and papers are accepted for publication on the understanding that they have not been published and are not being considered for publication elsewhere, and that they may not be re-published without sanction from the publishers. Contributions, which may be subject to editorial revision, should be condensed as far as possible and type-written on one side of the paper only, with double spacing and a one-inch margin on each side.

Each paper should conclude with a short summary, and references to previous work on the subject should be cited in the text with the author's name and year of publication in parentheses, e.g., Smith (1947); at the end of the paper a list should be given in alphabetical order according to the surname of the first author, and the titles of the journals should be abbreviated in accordance with the *World List of Scientific Periodicals*, e.g., *Brit. Vet. J.*, 107, 4.

Illustrations and Diagrams should, if possible, be larger than the size desired for reproduction. The areas to be reproduced in such illustrations (which the author considers to be essential and which are referred to specially in the text) should be outlined in pencil on the back of each. Legends should be typed on separate sheets of paper and not attached to the illustration. Photographs and photomicrographs should be printed on glossy paper. X-Ray films should not be submitted but prints of them, and preferable negative prints. Drawings and diagrams should be in black ink on Bristol board or stout white paper.

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